Tetra Tech EM Inc.

HEALTH AND SAFETY PLAN

Site Name: MichCon MGP	**Site Contact:** Ross Powers	**Telephone:**
Location: S. Green & S. Post Avenue, Detroit	**Client Contact:** Ross Powers	**Telephone:**
EPA I.D. No.	**Prepared By:** David Sawicki	**Date:**
Project No. G9009L0105017xxx	**Date of Proposed Activities:** 06/04/01 to ?	

Objectives:

Provide contractor oversight and documentation of field activities

Site Type: *Check as many as applicable.*

[X] Active	[ ] Confined space	[ ] Well field
[X] Inactive	[ ] Landfill	[ ] Unknown
[ ] Secure	[ ] Uncontrolled	[ ] Underground storage tank
[X] Unsecure	[X] Industrial	[ ] Other *(specify)*

Site Description and History:

The site was a former manufacture gas facility. All buildings have been razed. Some foundations may exist below grade. PRP has conducted investigations and documented contaminated soils from ground surface to 10 feet below grade. Soils will be removed and disposed of as a non-hazardous waste.

Note: A site map is provided on Page 9 of 12. Definitions and additional information about this form are provided on Page 12 of 12.

Waste Management Practices:

Contaminated soils will be excavated down to an approximate depth of 10 feet below grade by PRP.

Waste Types: ☐ Liquid ☒ Solid ☐ Sludge ☐ Gas ☐ Unknown

Waste Characteristics:

☐ Corrosive ☐ Flammable ☐ Radioactive
☒ Toxic ☒ Volatile ☐ Unknown
☐ Inert ☐ Reactive ☐ Other *(specify)* _____
☐ Ignitable

Hazards of Concern:

☒ Heat stress
☐ Cold stress
☐ Explosion or fire hazard
☐ Oxygen deficiency
☐ Radiological hazard
☒ Underground storage tanks
☐ Surface tanks
☒ Buried utilities
☐ Overhead utilities
☐ Biological hazard
☐ Noise
☐ Inorganic chemicals
☒ Organic chemicals
☒ Heavy equipment
☒ Other *(specify)* site is located in a high crime area

Explosion or Fire Potential: ☐ High ☐ Medium ☒ Low ☐ Unknown

Chemical Products Tetra Tech EM Inc. Will Use or Store On Site: (Attach a Material Safety Data Sheet [MSDS] for each item.)

- ☐ Alconox® or Liquinox®
- ☐ Hydrochloric acid (HCl)
- ☐ Nitric acid (HNO_3)
- ☐ Sodium hydroxide (NaOH)
- ☐ Sulfuric acid (H_2SO_4)
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____
- ☐ Other *(specify)* _____

Chemicals Present at Site	Highest Observed Concentration (specify units and media)	PEL/TLV (specify ppm or mg/m³)	IDLH Level (specify ppm or mg/m³)	Symptoms and Effects of Acute Exposure	Photo-ionization Potential (eV)
BTEX	unknown	1 ppm (benzene)	500 ppm (benzene)	irrit. Eyes, skin nose; respiritory sys, giddiness, headaches, nausea, staggered gait, fatigue,	Varies (< 10.2 v)
PAHs	Unknown	5 mg/m3 (mineral oil containg PAH)-TWA		Difficult to detect;	Varies
Cyanide	unknown	5 mg/m3 of air-TWA		heart pains, , vomiting, blood changes, headaches, deep breathing, shortness of breath, convulsions Skin- irritation and sores; Almond like odor on breath	NA

Notes:

A = Air
CARC = Carcinogenic
eV = Electron volt
GW = Groundwater
IDLH = Immediately dangerous to life or health
mg/m³ = Milligram per cubic meter
NA = Not available
NE = None established

PEL = Permissible exposure limit
ppm = Part per million
S = Soil
SW = Surface water
TLV = Threshold limit value
U = Unknown

ToxFAQs

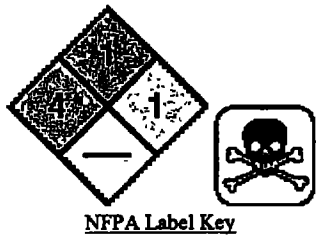
Cyanide

CAS# 57-12-5, 74-90-8, 143-33-9, 151-50-8, 592-01-8, 544-92-3, 506-61-6, 460-19-5, 506-77-4

September 1997

Potassium cyanide
KCN
Stereo Image
XYZ File

Search ChemFinder



NFPA Label Key

Vermont SIRI MSDS Archive

Agency for Toxic Substances and Disease Registry

This fact sheet answers the most frequently asked health questions (FAQs) about cyanide. For more information, call the ATSDR Information Center at 1-800-447-1544. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Cyanide is a very poisonous chemical. Exposure to high levels of cyanide harms the brain and heart, and may cause coma and death. Exposure to lower levels may result in breathing difficulties, heart pains, vomiting, blood changes, headaches, and enlargement of the thyroid gland. Cyanide has been found in at least 415 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is cyanide?

Cyanide is usually found joined with other chemicals to form compounds. Examples of simple cyanide compounds are hydrogen cyanide, sodium cyanide and potassium cyanide. Cyanide can be produced by certain bacteria, fungi, and algae, and it is found in a number of foods and plants. In the body, cyanide combines with a chemical to form Vitamin B_{12}. Cyanide occurs naturally in cassava roots, which are potato-like tubers of cassava plants grown in tropical countries.

Hydrogen cyanide is a colorless gas with a faint, bitter, almond-like odor. Sodium cyanide and potassium cyanide are both white solids with a bitter, almond-like odor in damp air. Cyanide and hydrogen cyanide are used in electroplating, metallurgy, production of chemicals, photographic development, making plastics, fumigating ships, and some mining processes.

What happens to cyanide when it enters the environment?

- Cyanide enters the environment from both natural processes and human industrial activities.
- In air, cyanide is mainly found as gaseous hydrogen cyanide; a small amount is present as fine dust particles
- It takes about 1–3 years for half of the hydrogen cyanide to disappear from the air
- Most cyanide in surface water will form hydrogen cyanide and evaporate

- Cyanide does not build up in the bodies of fish
- At high concentrations, cyanide becomes toxic to soil microorganisms and can pass through soil into underground water

How might I be exposed to cyanide?

- Breathing air, drinking water, touching soil, or eating foods containing cyanide
- Smoking cigarettes and breathing smoke-filled air during fires are major sources of cyanide exposure
- Breathing air near a hazardous waste site containing cyanide
- Eating foods containing cyanide compounds, such as cassava roots, lima beans, and almonds
- Working in an industry where cyanide is used or produced, such as electroplating, metallurgy, metal cleaning, and photography

How can cyanide affect my health?

Animal testing is sometimes necessary to find out how toxic substances might harm people or to treat those who have been exposed. Laws today protect the welfare of research animals and scientists must follow strict guidelines.

In large amounts, cyanide is very harmful to people. Exposure to high levels of cyanide in the air for a short time harms the brain and heart, and may cause coma and death.

Exposure to lower levels of cyanide for a long time may result in breathing difficulties, heart pains, vomiting, blood changes, headaches, and enlargement of the thyroid gland People who eat large amounts of cyanide may have symptoms including deep breathing and shortness of breath, convulsions, and loss of consciousness, and may die. Use of cassava roots as a primary food source in tropical Africa has led to high blood cyanide levels.

People with high blood cyanide levels have also shown harmful effects such as weakness of the fingers and toes, difficulty walking, dimness of vision, deafness, and decreased thyroid gland function, but chemicals other than cyanide may have contributed to these effects. Skin contact with cyanide can produce irritation and sores

It is not known whether cyanide can directly cause birth defects in people. Birth defects were seen in rats that ate diets of cassava roots. Effects on the reproductive system were seen in rats and mice that drank water containing sodium cyanide.

How likely is cyanide to cause cancer?

The **EPA** has determined that cyanide is not classifiable as to its human carcinogenicity There are no reports that cyanide can cause cancer in people or animals.

There are medical tests to measure blood and urine levels of cyanide; however, small amounts of cyanide are always detectable in blood and urine. Tissue levels of cyanide can be measured if cyanide poisoning is suspected, but cyanide is rapidly cleared from the body, so the tests must be done soon after the exposure. An almond-like odor in the breath may alert a doctor that a person was exposed to cyanide

Has the federal government made recommendations to protect human health?

The **EPA** has set a maximum contaminant level of cyanide in drinking water of 0.2 milligrams cyanide per liter of water (0.2 mg/L). The **EPA** requires that spills or accidental releases into the environment of 1 pound or more of hydrogen cyanide, potassium cyanide, sodium cyanide, calcium cyanide or copper cyanide be reported to the **EPA**.

The **Occupational Safety and Health Administration (OSHA)** and the **American Conference of Governmental Industrial Hygienists (ACGIH)** have set a permissible exposure limit of 5 milligrams of cyanide per cubic meter of air (5 mg/m^3) in the workplace during an 8-hour workday, 40-hour workweek.

Glossary

Carcinogenicity:
: Ability to cause cancer

CAS:
: Chemical Abstracts Service

Milligram (mg):
: One thousandth of a gram

PPM:
: Parts per million

Reference

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for cyanide (update). Atlanta, GA; U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

For more information, contact:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop E-29
Atlanta, GA 30333
Phone: 1-800-447-1544
Fax: 404-639-6359

U.S. Department of Health and Human Services
Public Health Service
Agency for Toxic Substances and Disease Registry

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ToxFAQs

Polycyclic Aromatic Hydrocarbons (PAHs)

CAS# 130498-29-2

September 1996

Polycyclic Aromatic Hydrocarbons
There is no molecular representation since this substance is a mixture of many compounds.

Agency for Toxic Substances and Disease Registry

This fact sheet answers the most frequently asked health questions about polycyclic aromatic hydrocarbons. For more information, you may call the ATSDR Information Center at 1-800-447-1544. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because these substances may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to polycyclic aromatic hydrocarbons usually occurs by breathing air contaminated by wild fires or coal tar, or by eating foods that have been grilled. PAHs have been found in at least 600 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are polycyclic aromatic hydrocarbons?

Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot

Some PAHs are manufactured. These pure PAHs usually exist as colorless, white, or pale yellow-green solids. PAHs are found in coal tar, crude oil, creosote, and roofing tar, but a few are used in medicines or to make dyes, plastics, and pesticides

What happens to PAHs when they enter the environment?

- PAHs enter the air mostly as releases from volcanoes, forest fires, burning coal, and automobile exhaust.
- PAHs can occur in air attached to dust particles.
- Some PAH particles can readily evaporate into the air from soil or surface waters.
- PAHs can break down by reacting with sunlight and other chemicals in the air, over a period of days to weeks.
- PAHs enter water through discharges from industrial and wastewater treatment plants
- Most PAHs do not dissolve easily in water. They stick to solid particles and settle to the bottoms of lakes or rivers
- Microorganisms can break down PAHs in soil or water after a period of weeks to months.
- In soils, PAHs are most likely to stick tightly to particles; certain PAHs move through soil to contaminate underground water.
- PAH contents of plants and animals may be much higher than PAH contents of soil or water in which they live.

How might I be exposed to PAHs?

- Breathing air containing PAHs in the workplace of coking, coal-tar, and asphalt production plants; smokehouses; and municipal trash incineration facilities.
- Breathing air containing PAHs from cigarette smoke, wood smoke, vehicle exhausts, asphalt roads, or agricultural burn smoke.
- Coming in contact with air, water, or soil near hazardous waste sites.
- Eating grilled or charred meats; contaminated cereals, flour, bread, vegetables, fruits, meats; and processed or pickled foods.
- Drinking contaminated water or cow's milk.
- Nursing infants of mothers living near hazardous waste sites may be exposed to PAHs through their mother's milk.

How can PAHs affect my health?

Mice that were fed **high levels** of one PAH during pregnancy had difficulty reproducing and so did their offspring. These offspring also had higher rates of birth defects and lower body weights. It is not known whether these effects occur in people.

Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both **short-** and **long-term** exposure. But these effects have not been seen in people.

How likely are PAHs to cause cancer?

The **Department of Health and Human Services (DHHS)** has determined that some PAHs may reasonably be expected to be carcinogens.

Some people who have breathed or touched mixtures of PAHs and other chemicals for **long periods** of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

Is there a medical test to show whether I've been exposed to PAHs?

In the body, PAHs are changed into chemicals that can attach to substances within the body. There are special tests that can detect PAHs attached to these substances in body tissues or blood. However, these tests cannot tell whether any health effects will occur or find out the extent or source of your exposure to the PAHs. The tests aren't usually available in your doctor's office because special equipment is needed to conduct them.

Has the federal government made recommendations to protect human health?

The **Occupational Safety and Health Administration (OSHA)** has set a limit of 0.2 milligrams of PAHs per cubic meter of air (0.2 mg/m^3). The **OSHA** Permissible Exposure Limit (PEL) for mineral oil mist that contains PAHs is 5 mg/m^3 averaged over an 8-hour exposure period.

The **National Institute for Occupational Safety and Health (NIOSH)** recommends that the average workplace air levels for coal tar products not exceed 0.1 mg/m^3 for a 10-hour workday, within a 40-hour workweek. There are other limits for workplace exposure for things that contain PAHs, such as coal, coal tar, and mineral oil.

Glossary

Carcinogen:
A substance that can cause cancer

Ingest:
Take food or drink into your body.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA. U S. Department of Health and Human Services, Public Health Service.

Where can I get more information?

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

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Public Health Service
Agency for Toxic Substances and Disease Registry

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Chemicals of Interest

Polynuclear Aromatic Hydrocarbons

The chemicals that have received the greatest attention at MGP sites with regard to human and/or ecological risks include polynuclear aromatic hydrocarbons (PAHs), benzene and other mono-aromatic hydrocarbons (BTEX), and cyanide compounds. Other compounds may be important at some sites especially if the MGP site has been used for different purposes over the years.

Polynuclear aromatic hydrocarbons (PAHs), a suite of compounds composed of two or more fused aromatic rings, are ubiquitous compounds in the environment. PAHs have been identified in soil, surface water, groundwater, sediment, and air. PAHs are predominantly introduced to the environment through natural and anthropogenic combustion processes. PAHs are also a common constituent of fossil fuels, and are commonly associated with manufactured gas plant (MGP) sites as components of coal tars. There are many sources of PAHs in the environment, and all individuals are exposed on a daily basis to these compounds in food, air, and water (Menzie et al., 1992). The USEPA has identified 16 unsubstituted PAHs as priority pollutants.

PAHs are very hydrophobic organic compounds and are relatively insoluble in water (Schwarzenbach et al., 1993). They have a high affinity for organic matter and, when present in soil or sediments, tend to remain bound to particles and dissolve only slowly in water. Soil, sediment, and suspended particulate matter (in air and water) represent important media for the transport of these chemicals. Ingestion of contaminated sediment and soils and inhalation of fugitive dust, can be important exposure pathways for PAHs. In addition, because PAHs are very hydrophobic and can bioaccumulate in some living organisms to elevated levels, consumption of contaminated foodstuffs can also be a significant exposure pathway. The toxicity of PAHs has been studied extensively in laboratory animal studies and human epidemiological studies (U.S. Department of Health and Human Services, 1995). PAHs were first identified as occupational carcinogens after a correlation was made between high exposure levels to soot and tar and an increased incidence in skin cancer. Formation of PAH-induced cancers in laboratory animals is well documented.

Animal data indicate that PAHs are readily absorbed after exposure by inhalation or oral intake and distributed to many tissues in the body. However, intestinal absorption of PAHs is dependent upon the presence of bile in the stomach. PAHs are absorbed via dermal exposure as shown by both human and animal studies, although very little is distributed to tissues. Following absorption, metabolism via the cytochrome P-450 monooxygenase system can occur, thus transforming PAHs to more water-soluble forms which can be efficiently eliminated from the body. The non-metabolized PAHs are not believed to be carcinogenic. During the metabolic process, some PAHs are activated to their carcinogenic intermediates. These intermediates can then bind to cellular macromolecules such as DNA, RNA, and proteins; damage to DNA can lead to the formation of a potentially carcinogenic cell. For many of the PAHs, however, metabolic activity results in the detoxification of PAHs through conversion to water-soluble metabolites.

Within the large class of PAHs, many structure-activity relationship studies have been conducted to relate chemical structure to carcinogenic activity. Animal studies have also been designed to test the carcinogenicity of the environmentally relevant PAHs. Eight PAHs are typically considered as possible or probable carcinogens (U.S. Department of Health and Human Services, 1995):

Carcinogenic PAHs	Noncarcinogenic PAHs
Benz(a)anthracene	Acenaphthene
Benzo(a)pyrene	Acenaphthylene
Benzo(g,h,i)perylene	Anthracene
Benzo(b)fluoranthene	Fluoranthene
Benzo(k)fluoranthene	Fluorene
Chrysene	Naphthalene
Dibenz(a,h)anthracene	Phenanthrene
Indeno(1,2,3-c,d)pyrene	Pyrene

Little data, animal or human, are available on the noncarcinogenic toxicities of the PAHs, and virtually no data exists on the acute effects of these compounds. The noncarcinogenic effects caused by high concentrations of PAHs may include damage to proliferating tissues such as the intestinal epithelium, bone marrow, lymphoid organs, and testes.

BTEX

BTEX, which is the collective term for the volatile organic compounds benzene, toluene, ethylbenzene, and xylenes, is a constituent of gasoline and other petroleum products. These light aromatic hydrocarbons are common contaminants at Manufactured Gas Plant (MGP) sites.

BTEX compounds are volatile and relatively soluble in water (Schwarzenbach et al., 1993). These compounds are mobile in the subsurface environment due to their high aqueous solubilities, and surface spills can leach to groundwater where transport can occur. Ingestion of or dermal contact with contaminated groundwater and inhalation of vapors can be important exposure pathways for BTEX compounds.

Benzene--At low levels in groundwater or air, the main health issue associated with benzene is the potential risk of cancer. Benzene is a recognized human carcinogen (USEPA Group A) and, because of its status as a carcinogen, it tends to drive risk assessments where there are potential exposures to BTEX either through inhalation or ingestion of groundwater.

Benzene also has the potential to cause noncarcinogenic health effects. At high levels, vapors from free product gasoline (including BTEX compounds) can cause acute health effects including irritation of the eyes and skin and narcotic effects. These acute adverse effects are unlikely to occur as a result of exposure to low levels in groundwater or air but need to be considered when free product is encountered.

Toluene--At low concentrations in groundwater or air, the primary health effects of toluene are noncarcinogenic. This compound is not considered a carcinogen. As with benzene, high levels of exposure associated with the presence of free product can result in irritant and narcotic effects.

Ethylbenzene--At low levels in groundwater or air, the primary health effects of ethylbenzene are noncarcinogenic. At elevated levels in the vapor phase, ethylbenzene is an irritant to the eyes, skin, and mucous membranes. At high concentrations, it can cause central nervous system effects such as narcosis. It is not considered a human or animal carcinogen because of a lack of animal bioassay and human studies.

Xylenes--The main health effects of xylenes from chronic exposures at low concentrations are noncarcinogenic. The available data suggest that xylene toxicity may be more severe after inhalation exposure than after oral exposure. Xylenes are not considered carcinogens. At elevated levels in air, xylenes can irritate eyes and may cause narcotic effects.

Cyanide Compounds

Cyanide compounds are common contaminants in the soils of former MGP sites and are often the focus of regulatory efforts. Soils at MGP sites frequently contain elevated concentrations of cyanide complexes, in particular iron cyanide complexes from the on-site disposal of purifier or oxide box wastes. Scrubbers containing iron oxide particles were used to remove hydrogen cyanide and hydrogen sulfide gases released during the coal carbonization process. Total cyanides have been found to be present in oxide wastes at concentrations ranging from 1 to greater than 2% by weight (Theis et al., 1994).

The iron cyanide complexes, ferrocyanide (hexacyanoferrate II, [Fe(CN)6]-4) and ferricyanide (hexacyanoferrate III, [Fe(CN)6]-3), have been found to be dominant cyanide species in contaminated soils at former MGP sites. Theis et al. (1994) reported that iron cyanide complexes comprised over 97% of the total cyanide in oxide wastes from two MGP sites. Ferric ferrocyanide, Fe4 (Fe(CN)6)3, known as Prussian Blue, is typically present in oxide wastes at elevated concentrations and is responsible for the deep blue color often visible in MGP site soils. Other cyanide complexes detected at MGP sites include copper and nickel cyanide species (e.g., Cu(CN)32- and Ni(CN)42-).

The toxicity and environmental fate of cyanide compounds in soils are dependent upon their chemical speciation. Free cyanide species such as HCN and CN- are extremely toxic, while iron cyanide complexes appear to exhibit lower toxicities. Cyanide in its free form [HCN(aq) and CN-] can be volatile and is biodegradable. Because cyanide complexes tend to be ionic in nature and have the ability to precipitate as solids, interactions with soil solids play an important role in their fate and transport. Cyanide complexes can also be persistent in soil environments.

Metallocyanide complexes such as iron cyanide complexes can chemically decompose to release toxic free cyanides, but slow decomposition kinetics can cause iron cyanides to be long-lived and inert species in soil environments (Meeussen et al., 1992). Decomposition is accelerated in sunlight and ultraviolet light; the rate of free cyanide release from metallocyanide complexes has been observed to be elevated for low pH conditions, high temperatures, and low cyanide concentrations (Meeussen et al., 1992). Iron cyanide complexes can thus persist in soils but, because they are not thermodynamically stable species and can slowly decompose to more toxic forms, they are not environmentally benign compounds.

Questions or comments about this document, contact: gnnet@www.gri.org

BENZO(A)PYRENE
CASRN: 50-32-8
For other data, click on the Table of Contents

Best Sections

Clean Water Act Requirements :

The attempt to develop a drinking water criterion for polynuclear aromatic hydrocarbons (PAH) as a class is hindered by several gaps in the scientific data base: (1) The PAH class is composed of numerous compounds having diverse biological effects and varying carcinogenic potential. A "representative" PAH mixture, has not been defined. (2) The common practice of using data derived from studies with benzo(a)pyrene to make generalizations concerning the effects of environmental PAH may not be scientifically sound. (3) No chronic animal toxicity studies involving oral exposure to PAH mixtures exist. (4) No direct human data concerning the effects of exposure to defined PAH mixtures exist. /Polynuclear aromatic hydrocarbons/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons (Draft) p.C-118 (1980)]
QC REVIEWED

Human Toxicity Excerpts :

Workers in coke oven plants have a higher incidence of lung cancer than the general population. They are exposed to a variety of chemicals, in particular the polycyclic aromatic hydrocarbons (PAH), including benzo(a)pyrene To evaluate the genotoxic effects of PAH exposure, air samples and urine samples were analyzed for PAH by capillary gas chromatography and high-performance liquid chromatography, respectively. Since benzo(a)pyrene is activated to 7-beta,8-alpha-dihydroxy-(9- alpha,10-alpha)-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (BPDE) and binds to DNA, we have used ultrasensitive enzymatic radioimmunoassay and synchronous fluorescence spectrophotometry to measure BPDE-DNA adducts in lymphocyte DNA. The mean PAH exposure levels are reduced 60% when the workers wore masks during work. When compared to exposure levels, the urinary excretion of PAH was relatively low. Approximately one-third of the workers had detectable putative BPDE-DNA adducts in lymphocytes by ultrasensitive enzymatic radioimmunoassay, and 10% of the samples had emission peaks at 379 nm by synchronous fluorescence spectrophotometry. The four most positive samples were the same in both of the assays. Antibodies to an epitope(s) on BPDE-DNA were found in the sera of approximately one-third of the workers. Detection of DNA adducts and antibodies to these adducts are internal indicators of exposure to benzo(a)pyrene.
[Haugen A et al; Cancer Res 46 (8): 4178-83 (1986)]**PEER REVIEWED**

Environmental Bioconcentration :

POLYCYCLIC AROMATIC HYDROCARBONS (PAH) WERE ANALYZED IN SURFACIAL SEDIMENTS & BENTHIC ORGANISMS IN SOUTHEASTERN LAKE ERIE, NEAR A LARGE COAL-FIRED POWER PLANT. SEDIMENT CONCN (530-770 PPB PAH) WERE RELATIVELY HOMOGENOUS THROUGHOUT MOST OF THE 150 SQUARE KM AREA, ALTHOUGH RIVER & NEARSHORE CONCENTRATIONS REACHED 4 PPM. OLIGOCHAETE WORMS DID NOT BIOCONCENTRATE (ON WET WT BASIS) ANY OF THE PAH. CHIRONOMIDE MIDGES COLLECTED 1 KM OFFSHORE EXHIBITED BIOCONCENTRATION OF 5 PAH ONE OF WHICH WAS PYRENE. FURTHER OFFSHORE, THESE APPARENT BIOCONCENTRATIONS DISAPPEARED, WITH MIDGES AT NEAR EQUILIBRIUM WITH SEDIMENTS. /PAH/
[EADIE BJ ET AL; CHEMOSPHERE 11 (2): 185-92 (1982)]**PEER REVIEWED**

Analytic Laboratory Methods :

AN INTEGRATED APPROACH COMPRISING A COMBINATION OF GLASS CAPILLARY GC, MASS SPECTROMETRY, LIQ CHROMATOGRAPHY & UV SPECTROMETRY WAS USED FOR UNAMBIGUOUS IDENTIFICATION OF POLYNUCLEAR AROMATIC HYDROCARBON (PAH) IN AIRBORNE PARTICULATES. LIQUID CHROMATOGRAPHY WITH ON-LINE UV SPECTRAL SCANNING WAS VALUABLE FOR DIFFERENTIATION OF ISOMERIC & COELUTING PAH. THE ADVANTAGES OF THIS APPROACH OVER GC/MS ALONE WERE ILLUSTRATED. PARENT PAH CONTAINING 3-7 RINGS WERE FOUND IN MOST SAMPLES EXAMINED; SOME ALKYL- & ALKOXY-PAH WERE ALSO DETECTED. A SIMPLE, 1-STEP PROCEDURE FOR ISOLATION OF PAH BY PREPARATIVE TLC IS ALSO REPORTED. /POLYNUCLEAR AROMATIC HYDROCARBONS/
[CHOUDHURY DR, BUSH B; ANAL CHEM 53 (9): 1351-6 (1981)]**PEER REVIEWED**

Analytic Laboratory Methods :

A simple, rapid method was developed for the separation and determination of polynuclear aromatic hydrocarbons (PAH) in barley malt. An ultrasonic-cyclohexane extraction method was used to separate the PAH from ground barley malt. The cyclohexane extracts were purified by chromatography through a water-deactivated silica gel-alumina column. The eluate from the column was concentrated and purified further by partitioning between dimethyl sulfoxide (DMSO) and cyclohexane. The DMSO extract was diluted with water and the PAH were extracted back into cyclohexane. The cyclohexane extract was washed with water, dried through sodium sulfate, evaporated and the resulting residue was dissolved in 80% aqueous acetonitrile:methanol (1:1) and subjected to reverse phase high performance liquid chromatography. Thirty barley malt samples were analyzed Peaks having the same retention time as the carcinogen benzo(a)pyrene were

isolated from 18 samples, and were equivalent to trace levels ranging from < 0 1-0.2 ppb Average recoveries of 11 PAH, including benzo (a)pyrene, benzo(b)fluoranthene, indeno(1,2,3-c)pyrene, and benz(a)anthracene, added to 25 g samples at 2.5 and 5 ppb, ranged from 78-97%, with a mean relative standard deviation of 6.6%.
[Joe FL et al; J Assoc Off Analyt Chem 65 (6): 1395-402 (1982)]**PEER REVIEWED**

Other Chemical/Physical Properties :

Adsorption and fluorescent spectra, fluorescent quantum yields, decay times, and O quenching constants of benzo(a)pyrene and benzo(e) pyrene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, indleno(1,2,3-cd)pyrene, benzo(a)anthracene, and cyclopenta (cd)pyrene, and of other airborne polycyclic aromatic hydrocarbons (PAH) were measured with and without oxygen in heptane at room temperature. .. This data can be used for optimal analysis of PAH by fluorescence spectrometry. The differences in the oxygen quenching of the fluorescent state of the various PAH can be used to analyze PAH mixtures which are difficult to separate by chromatographic techniques.
[Heinrich G, Guesten H; Polynucl Aromat Hydrocarbons: Chem Biol Eff Int Symp 4th 983-1003 (1980)]**PEER REVIEWED**

Absorption, Distribution & Excretion :

Polynuclear aromatic hydrocarbons (PAH), some of which are potent carcinogens, are common environmental pollutants. The transport processes for these hydrophobic compounds into cells and between intracellular membranes are diverse and are not well understood A common mechanism of transport is by spontaneous desorption and transfer through the aqueous phase From the partitioning parameters, we have inferred that the rate limiting step involves solvation of the transfer species in the interfacial water at the phospholipid surface. Transfer of 10 PAH out of phosphatidylcholine vesicles has been examined .. Results show that the molecular volume of the PAH is a rate-determining factor. Morever, high performance liquid chromatography (HPLC) data confirms the hypothesis that the rate of transfer is correlated with the size of the molecule and with the partitioning of the molecule between a polar and hydrocarbon phase. The kinetics and characteristics of the spontaneous transfer of carcinogens are likely to have a major impact on the competitive processes of PAH metabolism within cells /Polynuclear aromatic hyrocarbons/
[Plant AL et al; Chem-Biol Interact 44 (3): 237-46 (1983)]**PEER REVIEWED**

Analytic Laboratory Methods :

Nine polycyclic aromatic hydrocarbons (PHAs) contained in air samples collected on quartz fiber filters inside an urban tunnel and in a nearby mixed commerical residential area in the city of Rio de Janeiro, Brazil, were exposed to scrubbed air (to measure desorption loss) and the particle-free ambient air (to measure chemical reaction losses in the absence of desorption). The exposures were conducted for 5.5 to 9 hour periods at ambient temperature (22-26 deg C) at face velocities typical of high volume sampling. Under prevailing atmospheric conditions all nine PAHs experienced filter losses which (for most of them) followed first order kinetics For the ambient samples, in a 6 hour exposure period, the following five PAHs showed filter losses (% in parantheses) attributed exclusively to chemical reaction: benzo (b)fluoranthene (43), benzo(k)fluoranthene (39), benzo(a)pyrene (70), benzo(ghi)perylene (44), and indeno (1,2,3-cd)pyrene (41). The other four showed the following unassigned losses· pyrene (100), fluoranthene (65), crysene (72), and benzo(a)anthracene (71). The results are discussed in the light of possible filter artifacts in PAH sampling and the use of PAH profile signatures for source identification of atmospheric particulate matter in receptor modeling.
[Miguel AH et al; Int J Environ Anal Chem 26 (3-4): 265-78 (1986)]**PEER REVIEWED**

Probable Routes of Human Exposure :

. Finished waters from various treatment sites are transported to consumers through a variety of pipelines PAH's /polynuclear aromatic hydrocarbons/ leach from the tar or asphalt linings of these pipes ... resulting in increased concn of these cmpd in water reaching the consumers. ... Cement-lined pipes produce lower PAH concn, possibly because PAH's are adsorbed from water.
[National Research Council. Drinking Water & Health, Volume 4. Washington, DC: National Academy Press, 1981. 256]**PEER REVIEWED**

Analytic Laboratory Methods :

NIOSH Method 184. Polynuclear aromatic hydrocarbon (PAH) compounds are filtered from air with a glass fiber filter & collected on a silver membrane filter. The samples collected on the membrane filter are extracted with benzene. They are separated on an alumina column with n-pentane-diethyl ether mixtures as eluting solvents. The collected fractions are analyzed by a Recording UV Spectrophotometer, Cary Model 14, Varian Associates, or equivalent. The method is sensitive & capable of measuring several (PAH) in a single filter. Any substance that hinders the chromatographic separation or that absorbs at the same wavelength as the sample compound may interfere Benzo(a)pyrene is the 6th chem to elute among 9 specific PAHs. Its wavelength of maximum absorption is 382 NM. It has a baseline or background wavelength of 375, 390 NM A normal sample size corresponds to 2 to 3 mg of benzene-soluble material. Precision is not determined.
[U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual ofAnalytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1977-present.,p. V1 184-1]**PEER REVIEWED**

Analytic Laboratory Methods :

Contents of polycyclic aromatic hydrocarbons (PAHs) in the ointment and cosmetic bases liquid paraffin, yellow petrolatum, ichthammol and citric acid were determined by UV spectrophotometry (Japanese Pharmacopeia X), HPLC with fluorescence detection and TLC with fluorescence detection. The UV method gave poor results due to its low sensitivity and selectivity, whereas the HPLC method gave a good selectivity and quant results. TLC had an ability to detect PAHs and may be used in the detn of the PAH limit Benzo(a)pyrene, benzo(e) pyrene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(g,h,i)perylene and dibenz(a,h)anthracene were nondetectable in liquid paraffin and citric acid, and benzo(a)pyrene was not detected in ichthammol. Yellow petrolatum contained 0 2-1 75 ng/g benzo(a)pyrene in 3 samples analyzed.
[Kawamura T, Nakagawa T; Iyakuhin Kenkyu 16 (2): 336-42 (1985)]**PEER REVIEWED**

Human Toxicity Excerpts :

Polycyclic aromatic compounds (PAC) are ubiquitous pollutants in urban air that may pose risks to human health. In order to better assess the health risks associated with this class of compounds, a total of 67 polycyclic aromatic compounds that either have been identified (55) or are suspected to be present (12) in urban aerosol samples were tested for mutagenicity in a forward mutation assay based on human B-lymphoblastoid cells. The cell line used (designated hlAlv2) constitutively expresses the cytochrome p4501A1, which is known to be necessary for the metabolism of many promutagens. The polycyclic aromatic compounds tested included 39 polycyclic aromatic hydrocarbons (PAH), 19 oxygen-containing polycyclic aromatic hydrocarbons (oxy-PAH) and nine NO2-substituted polycyclic aromatic hydrocarbons (nitro-PAH). A total of 26 polycyclic aromatic hydrocarbons were mutagenic In comparing the minimum mutagenic concentrations of the mutagenic polycyclic aromatic hydrocarbons with that of benzo(a)pyrene (B[a]P) it was found that dibenzo(a,l)pyrene (DB(al)P), cyclopenta(c,d))pyrene (CPP), naphtho(2,1-a)pyrene, dibenzo(a,e)pyrene (DB(ae)P) and 1-methylbenzo(a)pyrene were 24 + or - 21, 6 9 + or - 4.2, 3.2 + or - 3.0, 2.9 + or - 2 9 and 1.6 + or - 1 4 times, respectively, more mutagenic than benzo(a)pyrene, and that dibenzo (a,k)fluoranthene and benzo(a)pyrene were approximately equally mutagenic. The 19 other mutagenic polycyclic aromatic hydrocarbons were between tested only phenalenone, 7H-benz(d,e)anthracen-7-one, 3-nitro-6H-dibenzo(b,d)pyran-6-one, cyclopenta(c,d)pyren-3(4H)-one, 6H-benzo(c,d)pyren-6-one (BPK) and anthanthrenequinone were mutagenic; however, with the exception of 6H-benzo(c,d)pyren-6-one, these were over 50 times less active than benzo(a)pyrene. 6H-benzo(c,d)pyren-6-one was benzo(a)pyrene Seven of the nitro-polycyclic aromatic hydrocarbons were mutagenic including 9-nitroanthracene, 1-nitrofluoranthene, 3-nitrofluoranthene, 1,3-dinitropyrene, 1,6-dinitropyrene (1,6-DNP) and 1,8-dinitropyrene 1,6-dinitropyrene wasnic nitro-polycyclic aromatic hydrocarbons were between 20 and 380 times less active than benzo(a)pyrene. These results are discussed in terms of their relevance for determining the most important mutagens in ambient air. Based on reported concentrations of polycyclic aromatic compounds in ambient aerosols, it is possible that cyclopenta(c,d))pyrene, dibenzo(a,e)pyrene, dibenzo(a,l)pyrene and 6H-benzo(c,d)pyren-6-one could account for a greater proportion of the mutagenicity than benzo(a)pyrene in some aerosols
[Durant JL et al; Mutation Research 371 (3-4): 123-57 (1996)]**PEER REVIEWED**

Analytic Laboratory Methods :

A TLC/HPLC (HIGH PRESSURE LIQUID CHROMATOGRAPHY) PROCEDURE FOR DETERMINATION OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH) OCCURRING IN ASPHALT FUMES (ADSORBED ON A PARTICULAR MATTER) IS DESCRIBED. THE METHOD IS BASED ON THE EXTRACTION OF ASPHALT FUME PARTICLES, COLLECTED ON GLASS-FIBER FILTERS, USING CARBON TETRACHLORIDE. A CLEAN UP STEP IS AIDED BY A TLC PROCEDURE ON ALUMINUM TRIOXIDE THINLAYER PLATES, USING A MIXTURE OF CYCLOHEXANE/ACETONE/ETHER AS THE MOBILE PHASE. UNDER UV-LIGHT, THE PAH ARE INDICATED AS FLUORESCENT SPOTS. SEPARATION OF THE COLLECTED PAH INTO INDIVIDUAL COMPONENTS & THEIR IDENTIFICATION IS PERFORMED BY THE AID OF A HPLC PROCEDURE
[RIETZ EB; ANAL LETT 12 (12): 143-54 (1979)]**PEER REVIEWED**

Cleanup Methods :

.. In surface waters, one-third of the total PAH is bound to larger suspended particles, a third is bound to finely dispersed particles, and the last third is present in dissolved form. The particle-bound portion of polycyclic aromatic hydrocarbons (PAH) can be removed by sedimentation, flocculation, and filtration processes. The remaining one-third dissolved PAH usually requires oxidation for partial removal/transformation. /polynuclear aromatic hydrocarbons/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons (Draft) p.C-4 (1980)]**PEER REVIEWED**

Cleanup Methods :

This method incorporates procedures for the destruction of laboratory wastes contaminated with PAH using an aqueous saturated potassium permanganate solution. This method has been tested for wastes contaminated with the following PAH. Benz(a)anthracene, 7,12-dimethylbenz(a)anthracene, benzo(a)pyrene, 3-methylcholanthrene, and 7-bromomethylbenz(a)anthracene. It has been studied collaboratively with a solution of benz(a)anthracene + benzo(a)pyrene + 7,12-dimenthylbenz(a)anthracene and a solution of 3-methylcholanthrene + dibenz(a,h)anthracene in cyclohexane. The method affords better than 95% destruction of all of the PAH tested except for dibenzo(a,h)anthracene, for which only variable and incomplete destruction could be achieved.
[Castegnaro, M., G. Grimmer, O. Hutzinger, W. Karcher, H. Kunte, M. LaFontaine, E.B. Sansone, G. Telling, and S.P. Tucker (eds.). Laboratory Decontaminationand Destruction of Carcinogens in Laboratory Wastes:

Some Polycyclic Aromatic Hydrocarbons. IARC Publications No. 49. Lyon, France: International Agency for Research on Cancer, 1983. 31]**PEER REVIEWED**

Human Toxicity Excerpts :

Previous studies in this laboratory have shown that polycyclic aromatic hydrocarbons (PAHs) alter Ca(2+) homeostasis and inhibit activation of both B and T lymphocytes obtained from rodents and humans. In the present studies, we demonstrate that a-naphthoflavone (ANF), an inhibitor of cytochrome p4501A activity, reduced the Ca(2+) elevation produced by benzo(a)pyrene in human peripheral blood mononuclear cell (HPBMC) lymphocytes. These results suggested that benzo(a)pyrene metabolites may play a role in intracellular Ca(2+) homeostasis in human lymphocytes. Reactive oxidative intermediates of benzo(a)pyrene produced in human peripheral blood mononuclear cell are known to be highly carcinogenic and have also been shown to be immunosuppressive. We examined the effects of benzo(a)pyrene (BaP), 7,12-dimethylbenz(a)anthracene (DMBA), benzo(e)pyrene (BeP), and anthracene, as well as certain benzo(a)pyrene metabolites, on the levels of intracellular Ca(2+) and glutathione in human peripheral blood mononuclear cell. While benzo(a)pyrene, 7,12-dimethylbenz (a)anthracene, benzo(e)pyrene, and anthracene did not cause a statistically significant decrease in GSH in human peripheral blood mononuclear cell at concentrations of 1 or 10 uM following a 6-, 48-, or 72-hr exposure, reactive benzo(a)pyrene metabolites including 4,5-epoxide benzo(a)pyrene and 7,8-diol-9,10-epoxide benzo(a)pyrene consistently produced a 20-30% depletion of glutathione in human peripheral blood mononuclear cell following a 6-hr treatment period. These benzo(a)pyrene metabolites also elevated intracellular Ca(2+) in human peripheral blood mononuclear cell during a 6-hr incubation. Results of these experiments suggest that metabolism of benzo(a) pyrene to certain epoxide metabolites lay be responsible for sulfhydryl damage leading to transient GSH depletion and Ca(2+) elevation. These results are consistent with the hypothesis that sulfhydryl damage by certain PAH metabolites may lead to altered Ca(2+) homeostasis, leading to inhibition of cell activation and proliferation in human peripheral blood mononuclear cell.
[Romero DL et al; Toxicol and Applied Pharmacol 144 (1): 62-9 (1997)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Buffalo river sediment extracts contained polynuclear aromatic hydrocarbons (PAH) which caused skin darkening, hyperplasia, skin papillomas, mild coarsening and local pigmentations in the brown bullhead (Ictalurus nebulosus) Sixteen PAH were identified in the sediment extract: fluorene, phenanthrene, anthracene, fluoranthene, 2-methylphenanthrene, pyrene, 2-methylanthracene, benzanthracene, chrysene, perylene, benzo(f)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, benzo(g,h,i)perylene, and indeno (1,2,3-c,d)pyrene.
[Black JJ; Polynucl Aromat Hydrocarbons Int Symp 7th 99-11 (1983)]**PEER REVIEWED**

Human Toxicity Excerpts :

The relative contributions of biologic and environmental factors on embryo-fetal development were elucidated in a population of pregnant women who were exposed to varying amounts of active cigarette smoke and women who were not exposed to cigarette smoke. The neonatal weight at birth, placental weight at delivery, duration of pregnancy, and placental xenobiotic (polynuclear aromatic hydrocarbon, PAH) metabolism potential were assessed in this population. The overall metabolic capability in exposed and unexposed placental tissue was measured by in vitro assays using microsomes and a polynuclear aromatic hydrocarbon substrate, benzo[a]pyrene (B[a]P). Toxicity potential was determined by benzo[a]pyrene-metabolite-DNA adduct generation under the same incubation condition. Cigarette smoke exposure increased the overall polynuclear aromatic hydrocarbon metabolism potential in placental tissues by approximately 200% (nonsmoker 176.2 + or - 33.6, n = 25; smoker 524.5 + or - 75.5, n = 32 pmol/mg protein) whereas polynuclear aromatic hydrocarbon-DNA adduct formation potential did not increase significantly over the basal level (nonsmoker 5002 + or - 830, n = 15; smoker 6172 + or - 1443, n = 22 fmol benzo[a]pyrene equivalent/umol DNA/mg protein). Exposure to cigarette smoke during pregnancy is deleterious to fetal development as reflected by reduced neonatal weight at birth. In contrast, placental weight reduction is indistinct, but placentae expressed markedly augmented overall xenobiotic (PAH) metabolism capability in response to cigarette smoke exposure during pregnancy, indicating placental metabolism may be an important mediator of adverse effects induced by such xenobiotic exposure.
[Sanyal MK et al; Reprod Toxicol 8 (5): 411-8 (1994)]**PEER REVIEWED**

Disposal Methods :

The method incorporates procedures for the destruction of laboratory wastes contaminated with PAH using concentrated sulfuric acid The method has been tested for wastes contaminated with the following PAH: Benz(a)anthracene, 7,12-dimethylbenz(a)anthracene, benzo(a) pyrene, 3-methylcholanthrene, 7-bromomethylbenz(a)anthracene, and dibenz(a,h)anthracene. It has been studied collaboratively using solutions of benz(a)anthracene + benzo(a)pyrene + 7,12-dimethylbenz(a)anthracene and solutions of methylcholanthrene + dibenz(a,h) anthracene in DMF and DMSO. ... The method affords better than 99% destruction in all solutions tested.
[Castegnaro, M., G. Grimmer, O. Hutzinger, W. Karcher, H. Kunte, M. LaFontaine, E.B. Sansone, G. Telling, and S.P. Tucker (eds.). Laboratory Decontaminationand Destruction of Carcinogens in Laboratory Wastes: Some Polycyclic Aromatic Hydrocarbons. IARC Publications No. 49. Lyon, France: International Agency for Research on Cancer, 1983. 25]**PEER REVIEWED**

Analytic Laboratory Methods :

A 4-STEP METHOD FOR THE REPRODUCIBLE ANALYSIS OF POLYNUCLEAR AROMATIC HYDROCARBONS (PAH) IN SMALL QUANTITIES OF CIGARETTE SMOKE CONDENSATE (CSC) IS PRESENTED. PAH WERE ISOLATED FROM AS

LITTLE AS 1 G OF CSC BY SOLVENT PARTITION, COLUMN CHROMATOGRAPHY, & ANALYSIS GEL FILTRATION (GF) THE GF ISOLATE WAS ANALYZED BY GAS CHROMATOGRAPHY. /POLYNUCLEAR AROMATIC HYDROCARBONS/
[SEVERSON RF ET AL; ANAL CHEM 48 (13): 1866 (1976)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Several well-documented examples of human exposure to carcinogens involve complex mixtures of polycyclic aromatic hydrocarbons (PAHs). Although the biological properties of many pure polycyclic aromatic hydrocarbons have been investigated, less is known about their effects when present as components of mixtures. As the ability to form DNA adducts in vivo is generally indicative of carcinogenic activity of polycyclic aromatic hydrocarbons, we have compared the DNA binding potencies of dibenzo(a,e)pyrene (DB(a,e)P), dibenzo (a,h)pyrene (DB(a,h)P), dibenzo(a,i)pyrene (DB(a,i)P), dibenzo(a,l)pyrene (DB(a,l)P) and benzo(a)pyrene (B(a)P), when applied topically, either singly or in combination, to the skin of male Parkes mice. DNA isolated from the skin and lungs was analyzed by 32P-postlabelling. The adducts formed by each polycyclic aromatic hydrocarbon exhibited markedly different chromatographic mobilities on polyethyleneimine-cellulose TLC plates. The relative binding potencies of the compounds in both skin and lungs were: dibenzo(a,l)pyrene > dibenzo(a,i)pyrene > dibenzo(a,e)pyrene, in good agreement with their reported carcinogenicities in mouse skin. The majority of adducts were removed from DNA within 21 days of treatment, but low levels of adducts were found to persist for at least 3 months in both tissues. When dibenzo(a,l)pyrene, dibenzo(a,e)pyrene and benzo(a)pyrene were applied together to mouse skin, a total binding 31% lower than expected was detected, while with a mixture of dibenzo(a,e)pyrene and benzo(a)pyrene the binding to DNA in skin was 65% higher than expected from the binding levels of the carcinogenes when applied singly. Other binary combinations of these three polycyclic aromatic hydrocarbons gave adduct levels similar to the sum of the binding levels of the individual components when applied singly. The results demonstrate the usefulness of 32P-post-labelling for the assessment of the DNA binding potencies of polycyclic aromatic hydrocarbons in mouse tissues, and for the detection of interactions between components of mixtures of carcinogens.
[Hughes NC, Phillips DH; Carcinogenesis (EYNSHAM); 11 (9): 1611-20 (1990)]**PEER REVIEWED**

Interactions :

Iron oxides are present in many occupational atmospheres mainly in iron ore mines and in steel industry Among these workers, epidemiological studies indicated an excess of lung cancer deaths. In mines, it was difficult to involve iron oxides exposure because there are other possible causes as radon, polycyclic aromatic hydrocarbon (PAH) present in diesel exhausts, silicosis or siderosis. The contradictory results of these studies are due to the differences of exposure levels or to the presence or not of these cofactors or of a sufficient prevention. But generally the results agree with an interaction of iron oxide dusts and smoking habits. It is unclear if this interaction supports an additive or multiplicative risk of lung cancer. Experimental studies with Fe203 showed that these particles are able to induce lung cancers only in the presence of polycyclic aromatic hydrocarbon when administered to animals. In vitro studies permitted to observe an interaction in the metabolism of benzo(a)pyrene (BaP) leading to a higher level of precursors of the ultimate carcinogen. As this metabolism of benzo(a)pyrene is known to be enhanced during lipoperoxidation, it is possible to involve this mechanism with Fe203. After phagocytosis and dissolution with production of ferric ions, Fe203 can enhance the production of reactive oxygen species responsible of damaging 60th lipidic constituents and DNA. Fe304 and mainly FeO may be more toxic, introducing directly ferrous ions in the cells after dissolution, but the canerogenicity of these compounds is unknown, making necessary to develop research.
[Haguenoer JM et al; Cent Eur J Public Health (4): 41-5 (1996)]**PEER REVIEWED**

Analytic Laboratory Methods :

OSW Method 8275A. Semivolatile Organic Compounds (PAHs and PCBs) in Soils/Sludges and Solid Wastes Using Thermal Extraction/Gas Chromatography/Mass Spectrometry (TE/GC/MS), soil/waste, TEGCMS.
[USEPA; EMMI. Environmental Monitoring Methods Index. Version 2.0 NTIS PB-95-502415 (1995)]**PEER REVIEWED**

Human Toxicity Excerpts :

Exposures to other chemical mixtures that contain PAHs, such as cigarette smoke, coal tar, coal tar pitch, and bitumens, have been associated with increased incidences of lung cancer in humans. /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Human Toxicity Excerpts :

Workers exposed to creosote containing numerous PAHs developed skin tumors .. /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Human Toxicity Excerpts :

Mortality studies have demonstrated that exposure to coke oven emissions, which contain a variety of PAHs, caused increased incidences of lung and genitourinary cancer mortality in coke oven workers ... . /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Interactions :

Benzo[a]pyrene (B[a]P) is able to inhibit the mutagenicity of 1-nitropyrene (1-NP) through the reduction of nitroreductase activity and formation of adducts with DNA. The relationships between the chemical structure of 9 polycyclic aromatic hydrocarbons (PAHs) and antagonistic effects on the 1-nitropyrene-induced mutation were evaluated by the binary mixtures of 1-nitropyrene and polycyclic aromatic hydrocarbons with Salmonella typhimurium TA98 in the absence of S9 mix. Remarkably different antagonistic effects of 9 polycyclic aromatic hydrocarbons on the mutagenicity of 1-nitropyrene were observed. Among the tested polycyclic aromatic hydrocarbons, coronene demonstrates the most antagonistic potential followed by benzo[g,h,i]perylene (B[g,h,i]P), benzo[e]pyrene (B[e]P), dibenzo[a,h]pyrene (DB[a,h]P), benzo[a]pyrene (B[a]P) and pyrene. Naphthalene, anthracene, and chrysene had only minor inhibitory activity on the 1-nitropyrene mutagenicity. The modifying effects of polycyclic aromatic hydrocarbons on the nitroreductase activity of TA98 strains in the presence of 1-nitropyrene were further examined from the production of 1-AP The statistical analytical data showed that the inhibitory effect of polycyclic aromatic hydrocarbons on the mutagenicity of 1-nitropyrene significantly correlated with their effects on the nitroreductase activity ($r = -0.69$, $p < 0.05$). In addition, the formation of 1-nitropyrene-DNA adducts of the binary mixtures of 1-nitropyrene and polycyclic aromatic hydrocarbon was determined by the 32P-postlabeling method. The results indicated that the modulatory effects of polycyclic aromatic hydrocarbons on the formation of 1-nitropyrene-DNA adducts were correlated well with their antagonistic activity ($r = -0.91$, $P < 0.011$. From the above results, the relationships between the chemical structure of polycyclic aromatic hydrocarbons and the antagonistic effects on the 1-nitropyrene mutagenicity were revealed by the surface area and electronic parameters of polycyclic aromatic hydrocarbons. The planar molecular area of polycyclic aromatic hydrocarbons was more convincingly correlated with the antagonistic effect on the mutagenicity of 1-nitropyrene ($r = -0.81$, $p < 0.01$) than that with the difference in energy, delta E, between EHOMO and ELUMO ($r = 0.69$, $p < 0.05$). According to the above, two possible mechanisms are involved in the interactive effect of the binary mixtures: (1) a higher binding affinity with nitroreductase for polycyclic aromatic hydrocarbons having a large planar surface area; and (2) a high energy of interaction between 1-nitropyrene and polycyclic aromatic hydrocarbons with a low delta E might decrease the nitroreductive capability.
[Cherng SH et al; H Mutat Res 367 (4): 177-85 (1996)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Rats and mice were exposed to combustion gases of coal-burning furnace enriched with benzo(a)pyrene (50-90 ug/cu m) and other polycyclic aromatic hydrocarbons (PAH) 16 hr/day, 5 days/wk. After approx 22-mo exposure, the incidence of lung neoplasm was approx 10-fold above controls.
[Heinrich U et al; Exp Pathol 29 (1): 29-34 (1986)]**PEER REVIEWED**

Interactions :

Several well-documented examples of human exposure to carcinogens involve complex mixtures of polycyclic aromatic hydrocarbons (PAHs). Although the biological properties of many pure polycyclic aromatic hydrocarbons have been investigated, less is known about their effects when present as components of mixtures. As the ability to form DNA adducts in vivo is generally indicative of carcinogenic activity of polycyclic aromatic hydrocarbons, we have compared the DNA binding potencies of dibenzo(a,e)pyrene (DB(a,e)P), dibenzo(a,h)pyrene (DB(a,h)P), dibenzo(a,i)pyrene (DB(a,i)P), dibenzo(a,l)pyrene (DB(a,l)P) and benzo(a)pyrene (B(a)P), when applied topically, either singly or in combination, to the skin of male Parkes mice. DNA isolated from the skin and lungs was analyzed by 32P-postlabelling The adducts formed by each polycyclic aromatic hydrocarbon exhibited markedly different chromatographic mobilities on polyethyleneimine-cellulose TLC plates. The relative binding potencies of the compounds in both skin and lungs were: dibenzo(a,l)pyrene > dibenzo(a,i)pyrene > dibenzo(a,e)pyrene, in good agreement with their reported carcinogenicities in mouse skin. The majority of adducts were removed from DNA within 21 days of treatment, but low levels of adducts were found to persist for at least 3 months in both tissues. When dibenzo(a,l)pyrene, dibenzo(a,e)pyrene and benzo(a)pyrene were applied together to mouse skin, a total binding 31% lower than expected was detected, while with a mixture of dibenzo(a,e)pyrene and benzo(a)pyrene the binding to DNA in skin was 65% higher than expected from the binding levels of the carcinogenes when applied singly. Other binary combinations of these three polycyclic aromatic hydrocarbons gave adduct levels similar to the sum of the binding levels of the individual components when applied singly. The results demonstrate the usefulness of 32P-post-labelling for the assessment of the DNA binding potencies of polycyclic aromatic hydrocarbons in mouse tissues, and for the detection of interactions between components of mixtures of carcinogens.
[Hughes NC, Phillips DH; Carcinogenesis (EYNSHAM); 11 (9): 1611-20 (1990)]**PEER REVIEWED**

Human Toxicity Excerpts :

Previous studies have shown that polycyclic aromatic hydrocarbons (PAHs) mobilize intracellular Ca2+ in human T cells by inositol trisphosphate-dependent mechanisms resulting from activation of phospholipase C-gamma by SRC-related protein tyrosine kinases, thereby mimicking antigen-receptor activation. Ca2+ appears to play an important second messenger role in growth factor control of cell proliferation in human mammary epithelial cells (HMEC) such as the epidermal growth factor receptor pathway. The purpose of the present studies was to determine if polycyclic aromatic hydrocarbons are able to increase intracellular Ca2+ in primary cultures of human mammary epithelial cells and increase cell proliferation. Two carcinogenic and two non-carcinogenic polycyclic aromatic hydrocarbons were tested for their ability to increase intracellular Ca2+ in human mammary epithelial cells. The carcinogenic polycyclic aromatic hydrocarbons dimethylbenz(a)anthracene (DMBA) and benzo[a] pyrene (BaP) were able to cause Ca2+ elevation in human mammary epithelial cells at early time points (2 hr) and caused sustained alterations in Ca2+ homeostasis (18 hr). Dimethylbenz(a)anthracene showed maximal effects at early time points (2 hr), while benzo(a)pyrene showed maximal effects on sustained Ca2- (18 hr). 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), a potent dioxin and tumor promoter, produced maximal Ca2+ elevation at 2 hr, with a return to near

baseline levels by 6 hr. The non-carcinogenic polycyclic aromatic hydrocarbons benzo[e]pyrene and anthracene did not significantly alter intracellular Ca2+ at any time point. alpha-Naphthoflavone significantly reduced the Ca2+ response induced by benzo(a)pyrene treatment, but not by dimethylbenz(a)anthracene or 2,3,7,8-Tetrachlorodibenzo-p-dioxin, suggesting that p450 1A or 1B metabolism of benzo(a)pyrene may be important in the sustained Ca2+ elevating response. In evaluating the effects of benzo(a)pyrene on human mammary epithelial cells proliferation, benzo(a)pyrene was found to increase the number of cells recovered after 4 days in culture in the absence or presence of various concentrations of epidermal growth factor. These studies provide initial evidence that Ca2+ signaling may be associated with mitogenesis in human mammary epithelial cells, which may play a role in tumor promotion and progression produced by polycyclic aromatic hydrocarbons.
[Tannheimer SL et al; Carcinogenesis 18 (6): 1177-82 (1997)]**PEER REVIEWED**

Human Toxicity Excerpts :

Dermal exposure to coal tar and shale oils containing PAHs have been associated with increased incidences of skin tumors in humans . /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Analytic Laboratory Methods :

The X-ray excited optical luminescence (XEOL) of a concentrate in n-heptane of the neutral fraction isolated from by-products of coal combustion and conversion, and from shale and fuel oils was utilized to obtain profiles of their PAH content. Components which should be unequivocally identified include benzo(a)anthracene, BaP, benzo(e)pyrene, benzo(ghi)perylene, pyrene, benzo(k)fluoranthene, and coronene.
[Fassel VA et al; Analytical Chem 52 (1): 159-64 (1980)]**PEER REVIEWED**

Analytic Laboratory Methods :

ULTRASONIC EXTRACTION OF AIRBORNE PARTICULATE MATERIAL ON HI-VOL FILTERS IS DESCRIBED. ALMOST ALL POLAR COMPOUNDS ARE REMOVED DURING EXTRACTION BY ADSORPTION ON THE SURFACE OF SHREDDED GLASS FIBERS & CONTROLLED PORE GLASS POWDER (CPG). NON-POLAR POLYNUCLEAR AROMATIC HYDROCARBONS (PAH) IN THE EXTRACTS ARE SEPARATED AT ROOM TEMP BY HIGH PRESSURE LIQUID CHROMATOGRAPHY (HPLC) ON REVERSE PHASE VYDAC USING ACETONITRILE:WATER (70-30 VOL/VOL) AS CHROMATOGRAPHIC SOLVENT. B(A)P ELUTES IN APPROX 14 MIN. PRECISION & ACCURACY MEASUREMENTS INDICATE FULL RECOVERY & GOOD EXTRACTION REPRODUCIBILITY. DETECTION LIMIT FOR B(A)P AT F 290/389 IS LESS THAN 100 PG. TOTAL ANALYSIS TIME IS APPROX 1.5 HR.
[GOLDEN C, SAWICKI E; ANAL LETT 11 (12): 1051-62 (1978)]**PEER REVIEWED**

Clean Water Act Requirements :

For the maximum protection of human health from the potential carcinogenic effects due to exposure of polynuclear aromatic hydrocarbons through ingestion of contaminated water and contaminated aquatic organisms, ... therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 1x10-5, 1x10-6, and 1x10-7. The corresponding criteria /for ambient water/ are 28.0 ng/l, 2.8 ng/l, and 0.28 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 344 0 ng/l, a 31.1 ng/l, and 3 11 ng/l respectively. /Polynuclear aromatic hydrocarbons based on benzo (a)pyrene as the model PAH/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons p.C-121 (1980)] **QC REVIEWED**

Non-Human Toxicity Excerpts :

In animal studies, exposure to high levels of diesel exhaust particulates overwhelms the normal clearance mechanisms and results in lung burdens of diesel exhaust particulates that exceed those predicted from observations at lower exposure concentrations. A variable amount of the mass of diesel exhaust particulates is extractable with strong organic solvents. The extracted material contains more than a thousand individual compounds and is mutagenic in a number of bacterial and mammalian cell assays. Bioassay-directed chemical analysis of diesel exhaust particulates had identified several hundred compounds. Many are PAHs, some of which are considered to have human carcinogenic potential. The association of benzo(a)pyrene and nitropyrene with diesel exhaust particulates prolongs their retention in the lungs.
[McClellan RD; Annu Rev Pharmacol Toxicol 27: 279-300 (1987)]**PEER REVIEWED**

Emergency Medical Treatment :

EMT Copyright Disclaimer:

Portions of the POISINDEX(R) database are provided here for general reference THE COMPLETE POISINDEX(R) DATABASE, AVAILABLE FROM MICROMEDEX, SHOULD BE CONSULTED FOR ASSISTANCE IN THE DIAGNOSIS OR TREATMENT OF

The following Overview, *** POLYNUCLEAR AROMATIC HYDROCARBONS ***, is relevant for this HSDB record chemical.

Life Support:

- This overview assumes that basic life support measures have been instituted.

Clinical Effects:

SUMMARY OF EXPOSURE

0.2.1.1 ACUTE EXPOSURE

- In general, **PAHs** have a low order of acute toxicity in humans.
- **PAHs** and other compounds found in COAL TAR can produce a variety of non-cancer effects with chronic exposure. Chronic effects include:
  1. EYES - Photosensitivity and irritation.
  2. RESPIRATORY - Irritation with cough and bronchitis.
  3. MOUTH - Leukoplakia.
  4. DERMAL - "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, dermal burns, photosensitivity, acneiform lesions, irritation.
  5. HEPATIC/RENAL - Mild hepatotoxicity or mild nephrotoxicity (animals).
  6. GENITOURINARY - Hematuria.
- CANCER is the most significant **PAH** toxicity endpoint.
  1. Increased incidences of cancers of the skin, bladder, lung and gastrointestinal tract have been described in **PAH**-exposed workers.

RESPIRATORY

0.2.6.1 ACUTE EXPOSURE

- Irritation, chronic cough, bronchitis, and bronchogenic cancer can occur with chronic exposure.

GASTROINTESTINAL

0.2.8.1 ACUTE EXPOSURE

- Leukoplakia and cancers of the lip and oral cavity can develop with chronic exposure.

HEPATIC

0.2.9.1 ACUTE EXPOSURE

- Mild hepatotoxicity has been reported in **PAH**-exposed rats.

GENITOURINARY

0.2.10.1 ACUTE EXPOSURE

- Hematuria, kidney and bladder cancer are possible effects of chronic exposure. Mild nephrotoxicity has been documented in **PAH**-exposed rats.

HEMATOLOGIC

0.2.13.1 ACUTE EXPOSURE

- Agranulocytosis, anemia, leukopenia, and pancytopenia developed in rats chronically fed **PAHs**.

DERMATOLOGIC

0.2.14.1 ACUTE EXPOSURE

- PRECANCEROUS LESIONS - "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, dermal burns, acneiform lesions, photosensitization and cancer may develop following chronic exposure.

IMMUNOLOGIC

0.2.19.1 ACUTE EXPOSURE

- An effect of **PAHs** on immune function might aid in the development of neoplasms. A number of **PAH** compounds are immunotoxic, and some suppress selective components of the immune system.

REPRODUCTIVE HAZARDS

- In experimental animal studies, **PAHs** and metabolites cross the placenta. Female offspring of experimental animals exposed to **PAHs** during pregnancy have a decrease in the number of functional oocytes, sometimes such that they are infertile.
- **PAHs** are lipophilic and are excreted in breast milk, allowing for secondary exposure of nursing infants, although the potential significance of such exposure has not been determined.

CARCINOGENICITY

0.2.21.1 IARC CATEGORY

- PNAs AS A GROUP -
  1. Varies. Classified as Group 2A - probably carcinogenic to humans, to Group 3 - not classifiable as to its carcinogenicity to humans (IARC, 1989).
- INDIVIDUAL PNAs -
  1. ANTHRACENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  2. BENZ[A]ANTHRACENE - 2A, probably carcinogenic to humans (IARC, 1987).
  3. BENZO[K]FLUORANTHENE - 2B, possibly carcinogenic to humans (IARC, 1987).
  4. BENZO[GHI]PERYLENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  5. BENZO[A]PYRENE - 2A - probably carcinogenic to humans (IARC, 1987).
  6. BENZO[E]PYRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  7. CHRYSENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  8. CORONENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  9. DIBENZ[A,H]ACRIDINE - 2B - possibly carcinogenic to humans (IARC, 1987).
  10. DIBENZ[A,H]ANTHRACENE - 2A - probably carcinogenic to humans (IARC, 1987).
  11. 7H-DIBENZO[C,G]CARBAZOLE - 2B - possibly carcinogenic to humans (IARC, 1987).
  12. PHENANTHRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  13. PYRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
- RELATED SUBSTANCES & PROCESSES -
  1. COAL GASIFICATION; COAL-TAR PITCHES; COAL-TARS - 1 - carcinogenic to humans (IARC, 1987).
  2. COKE PRODUCTION - 1 - carcinogenic to humans (IARC, 1987).
  3. CREOSOTES - 2A - probably carcinogenic to humans (IARC, 1987).
  4. UNTREATED AND MILDLY TREATED MINERAL OILS - 1 - carcinogenic to humans (IARC, 1989).
  5. HIGHLY REFINED MINERAL OILS - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1989).

0.2.21.2 HUMAN OVERVIEW

- Cancer is the most significant **PAH** toxicity endpoint. Some, but not all, **PAHs** are carcinogens. Certain **PAH** parent compounds are weak carcinogens, only becoming potent carcinogens after undergoing metabolism. Chronic or repeated exposure increases the likelihood of cancer initiation, as well as the potential for metabolism of a **PAH** procarcinogen to a carcinogen.
- Increased incidences of skin, bladder, lung, and possibly gastrointestinal tract cancers have been described in **PAH**-exposed workers, particularly associated with coal carbonization, coal gasification, and coke oven work.

0.2.21.3 ANIMAL OVERVIEW

- Lung tumors were induced in female mice from inhalation of exhausts containing **PAHs** (Schulte et al, 1994).

GENOTOXICITY

- CYTOGENETIC MARKERS OF HUMAN **PAH** EXPOSURE - Cells containing high frequencies of chromosomal aberrations were a sensitive marker for exposure to **PAHs** in coke oven and graphic electrode plant workers, compared with unexposed controls. There was no relation between cytogenetic findings and levels of benzo(a)pyrene-hemoglobin adducts (Buchet et al, 1995).
  1. Arylamines derived from carcinogenic **PAHs** are mutagenically activated through S9-mediated metabolism of the related amine (Fu et al, 1982).
  2. Nitro-**PAH** derivatives are potent bacterial mutagens. The mutagenic activity is dependent on enzymatic reduction of the nitro group and may require oxidative

metabolism (Grosovsky et al, 1999; Rosenkranz & Mermelstein, 1985).
3. In cultured human cells, benzo(a)pyrene and 7,12-demethylbenz(a)anthracene only caused a significant increase in T6 guanine-resistant mutations in the presence of cultured rat hepatocytes (Tong et al, 1981).
4. Nitrated **PAHs** have caused dose-dependent cell transformations in Syrian hamster embryo cells (DiPaolo et al, 1983). Metabolic reduction of the nitro- group on **PAHs** may be involved in their mutagenic effects.
5. Persons with a high degree of inducibility of the enzyme, aryl hydrocarbon hydroxylase, may be a high-risk population (ATSDR, 1993).

- MUTAGENIC INTERMEDIATES -

1. The **PAH** metabolic intermediates, diol-epoxides, are presumably mutagenic and can react to form DNA adducts, which may affect normal cellular replication (ATSDR, 1993; Ellenhorn & Barceloux, 1988; Pike, 1992; Amin et al, 1993; Ronai et al, 1994).

- LACK OF EFFECT

1. Amongst a small group of Swedish road pavers exposed to asphalt fumes, there were no significant increases in sister chromatic exhanges or micronuclei in peripheral blood/lymphocytes as compared to unexposed controls (Jarvholm et al, 1999).

Laboratory:

- **PAHs** have been determined in the blood and tissues of experimental animals. Direct biologic measurement of **PAHs** currently is not clinically useful or cost-effective. Indirect methods of determining exposure are available but have not yet proven clinically useful (ATSDR, 1990).
- Acute respiratory effects in persons at **PAH**-containing workplaces are typically due to other toxic agents at the worksite (ATSDR, 1990). Arterial blood gases, chest x-ray, and other monitoring may be indicated, based on the patient's presentation and the exposure characteristics.
- Chronic effects, particularly cancer, are more common than acute toxicity. Routine monitoring and physical assessments (e.g, complete blood count, hepatic and renal function tests, chest xray and pulmonary function tests, dermal assessments) of individuals with significant exposure is recommended, even in the absence of symptoms (ATSDR, 1990).

Treatment Overview:

ORAL EXPOSURE

- Toxicity from these substances involves chronic exposure, toxicity after acute ingestions is unlikely and gastric decontamination is generally NOT indicated.

INHALATION EXPOSURE

- DECONTAMINATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer 100 percent humidified supplemental oxygen with assisted ventilation as required.
- Inhalational exposure to **PAHs** may be complicated by exposure to other substances which produce acute respiratory and systemic effects. Treat according to clinical presentation and exposure history.

1. If bronchospasm and wheezing occur, consider treatment with inhaled sympathomimetic agents.
2. Carefully observe patients with inhalation exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.
3. Monitor arterial blood gases, pulmonary function, and chest x-ray for patients with significant exposure.

EYE EXPOSURE

- DECONTAMINATION: Exposed eyes should be irrigated with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.

DERMAL EXPOSURE

- DECONTAMINATION: Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists.
- Treat dermal irritation or burns with standard topical therapy. Patients developing dermal hypersensitivity reactions may require treatment with systemic or topical corticosteroids or antihistamines.
- Some chemicals can produce systemic poisoning by absorption through intact skin. Carefully observe patients with dermal exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.

Range of Toxicity:

- The minimum lethal human dose to this agent has not been delineated.
- The maximum tolerated human exposure to this agent has not been delineated.

[Rumack BH: POISINDEX(R) Information System. Micromedex, Inc., Englewood, CO, 2001; CCIS Volume 109, edition exp August, 2001. Hall AH & Rumack BH (Eds):TOMES(R) Information System. Micromedex, Inc., Englewood, CO, 2001; CCIS Volume 109, edition exp August, 2001.] **PEER REVIEWED**

BENZO(A)PYRENE
CASRN: 50-32-8
For other data, click on the Table of Contents

Best Sections

Clean Water Act Requirements :

The attempt to develop a drinking water criterion for polynuclear aromatic hydrocarbons (PAH) as a class is hindered by several gaps in the scientific data base (1) The PAH class is composed of numerous compounds having diverse biological effects and varying carcinogenic potential. A "representative" PAH mixture, has not been defined. (2) The common practice of using data derived from studies with benzo(a)pyrene to make generalizations concerning the effects of environmental PAH may not be scientifically sound. (3) No chronic animal toxicity studies involving oral exposure to PAH mixtures exist. (4) No direct human data concerning the effects of exposure to defined PAH mixtures exist. /Polynuclear aromatic hydrocarbons/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons (Draft) p.C-118 (1980)]
QC REVIEWED

Human Toxicity Excerpts :

Workers in coke oven plants have a higher incidence of lung cancer than the general population. They are exposed to a variety of chemicals, in particular the polycyclic aromatic hydrocarbons (PAH), including benzo(a)pyrene. To evaluate the genotoxic effects of PAH exposure, air samples and urine samples were analyzed for PAH by capillary gas chromatography and high-performance liquid chromatography, respectively Since benzo(a)pyrene is activated to 7-beta,8-alpha-dihydroxy-(9- alpha,10-alpha)-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (BPDE) and binds to DNA, we have used ultrasensitive enzymatic radioimmunoassay and synchronous fluorescence spectrophotometry to measure BPDE-DNA adducts in lymphocyte DNA. The mean PAH exposure levels are reduced 60% when the workers wore masks during work. When compared to exposure levels, the urinary excretion of PAH was relatively low. Approximately one-third of the workers had detectable putative BPDE-DNA adducts in lymphocytes by ultrasensitive enzymatic radioimmunoassay, and 10% of the samples had emission peaks at 379 nm by synchronous fluorescence spectrophotometry. The four most positive samples were the same in both of the assays. Antibodies to an epitope(s) on BPDE-DNA were found in the sera of approximately one-third of the workers. Detection of DNA adducts and antibodies to these adducts are internal indicators of exposure to benzo(a)pyrene.
[Haugen A et al; Cancer Res 46 (8): 4178-83 (1986)]**PEER REVIEWED**

Environmental Bioconcentration :

POLYCYCLIC AROMATIC HYDROCARBONS (PAH) WERE ANALYZED IN SURFACIAL SEDIMENTS & BENTHIC ORGANISMS IN SOUTHEASTERN LAKE ERIE, NEAR A LARGE COAL-FIRED POWER PLANT. SEDIMENT CONCN (530-770 PPB PAH) WERE RELATIVELY HOMOGENOUS THROUGHOUT MOST OF THE 150 SQUARE KM AREA, ALTHOUGH RIVER & NEARSHORE CONCENTRATIONS REACHED 4 PPM. OLIGOCHAETE WORMS DID NOT BIOCONCENTRATE (ON WET WT BASIS) ANY OF THE PAH. CHIRONOMIDE MIDGES COLLECTED 1 KM OFFSHORE EXHIBITED BIOCONCENTRATION OF 5 PAH ONE OF WHICH WAS PYRENE. FURTHER OFFSHORE, THESE APPARENT BIOCONCENTRATIONS DISAPPEARED, WITH MIDGES AT NEAR EQUILIBRIUM WITH SEDIMENTS. /PAH/
[EADIE BJ ET AL; CHEMOSPHERE 11 (2): 185-92 (1982)]**PEER REVIEWED**

Analytic Laboratory Methods :

AN INTEGRATED APPROACH COMPRISING A COMBINATION OF GLASS CAPILLARY GC, MASS SPECTROMETRY, LIQ CHROMATOGRAPHY & UV SPECTROMETRY WAS USED FOR UNAMBIGUOUS IDENTIFICATION OF POLYNUCLEAR AROMATIC HYDROCARBON (PAH) IN AIRBORNE PARTICULATES LIQUID CHROMATOGRAPHY WITH ON-LINE UV SPECTRAL SCANNING WAS VALUABLE FOR DIFFERENTIATION OF ISOMERIC & COELUTING PAH. THE ADVANTAGES OF THIS APPROACH OVER GC/MS ALONE WERE ILLUSTRATED. PARENT PAH CONTAINING 3-7 RINGS WERE FOUND IN MOST SAMPLES EXAMINED; SOME ALKYL- & ALKOXY-PAH WERE ALSO DETECTED. A SIMPLE, 1-STEP PROCEDURE FOR ISOLATION OF PAH BY PREPARATIVE TLC IS ALSO REPORTED /POLYNUCLEAR AROMATIC HYDROCARBONS/
[CHOUDHURY DR, BUSH B; ANAL CHEM 53 (9): 1351-6 (1981)]**PEER REVIEWED**

Analytic Laboratory Methods :

A simple, rapid method was developed for the separation and determination of polynuclear aromatic hydrocarbons (PAH) in barley malt An ultrasonic-cyclohexane extraction method was used to separate the PAH from ground barley malt. The cyclohexane extracts were purified by chromatography through a water-deactivated silica gel-alumina column. The eluate from the column was concentrated and purified further by partitioning between dimethyl sulfoxide (DMSO) and cyclohexane. The DMSO extract was diluted with water and the PAH were extracted back into cyclohexane. The cyclohexane extract was washed with water, dried through sodium sulfate, evaporated and the resulting residue was dissolved in 80% aqueous acetonitrile methanol (1.1) and subjected to reverse phase high performance liquid chromatography Thirty barley malt samples were analyzed. Peaks having the same retention time as the carcinogen benzo(a)pyrene were

isolated from 18 samples, and were equivalent to trace levels ranging from < 0.1-0.2 ppb Average recoveries of 11 PAH, including benzo (a)pyrene, benzo(b)fluoranthene, indeno(1,2,3-c)pyrene, and benz(a)anthracene, added to 25 g samples at 2.5 and 5 ppb, ranged from 78-97%, with a mean relative standard deviation of 6.6%.
[Joe FL et al; J Assoc Off Analyt Chem 65 (6): 1395-402 (1982)]**PEER REVIEWED**

Other Chemical/Physical Properties :

Adsorption and fluorescent spectra, fluorescent quantum yields, decay times, and O quenching constants of benzo(a)pyrene and benzo(e) pyrene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, indleno(1,2,3-cd)pyrene, benzo(a)anthracene, and cyclopenta (cd)pyrene, and of other airborne polycyclic aromatic hydrocarbons (PAH) were measured with and without oxygen in heptane at room temperature. .. This data can be used for optimal analysis of PAH by fluorescence spectrometry. The differences in the oxygen quenching of the fluorescent state of the various PAH can be used to analyze PAH mixtures which are difficult to separate by chromatographic techniques.
[Heinrich G, Guesten H; Polynucl Aromat Hydrocarbons: Chem Biol Eff Int Symp 4th 983-1003 (1980)]**PEER REVIEWED**

Absorption, Distribution & Excretion :

Polynuclear aromatic hydrocarbons (PAH), some of which are potent carcinogens, are common environmental pollutants. The transport processes for these hydrophobic compounds into cells and between intracellular membranes are diverse and are not well understood. A common mechanism of transport is by spontaneous desorption and transfer through the aqueous phase From the partitioning parameters, we have inferred that the rate limiting step involves solvation of the transfer species in the interfacial water at the phospholipid surface. Transfer of 10 PAH ... out of phosphatidylcholine vesicles has been examined. ... Results show that the molecular volume of the PAH is a rate-determining factor. Morever, high performance liquid chromatography (HPLC) data confirms the hypothesis that the rate of transfer is correlated with the size of the molecule and with the partitioning of the molecule between a polar and hydrocarbon phase. The kinetics and characteristics of the spontaneous transfer of carcinogens are likely to have a major impact on the competitive processes of PAH metabolism within cells /Polynuclear aromatic hyrocarbons/
[Plant AL et al; Chem-Biol Interact 44 (3): 237-46 (1983)]**PEER REVIEWED**

Analytic Laboratory Methods :

Nine polycyclic aromatic hydrocarbons (PHAs) contained in air samples collected on quartz fiber filters inside an urban tunnel and in a nearby mixed commerical residential area in the city of Rio de Janeiro, Brazil, were exposed to scrubbed air (to measure desorption loss) and the particle-free ambient air (to measure chemical reaction losses in the absence of desorption) The exposures were conducted for 5.5 to 9 hour periods at ambient temperature (22-26 deg C) at face velocities typical of high volume sampling Under prevailing atmospheric conditions all nine PAHs experienced filter losses which (for most of them) followed first order kinetics. For the ambient samples, in a 6 hour exposure period, the following five PAHs showed filter losses (% in parantheses) attributed exclusively to chemical reaction: benzo (b)fluoranthene (43), benzo(k)fluoranthene (39), benzo(a)pyrene (70), benzo(ghi)perylene (44), and indeno (1,2,3-cd)pyrene (41). The other four showed the following unassigned losses: pyrene (100), fluoranthene (65), crysene (72), and benzo(a)anthracene (71). The results are discussed in the light of possible filter artifacts in PAH sampling and the use of PAH profile signatures for source identification of atmospheric particulate matter in receptor modeling.
[Miguel AH et al; Int J Environ Anal Chem 26 (3-4): 265-78 (1986)]**PEER REVIEWED**

Probable Routes of Human Exposure :

.. Finished waters from various treatment sites are transported to consumers through a variety of pipelines. PAH's /polynuclear aromatic hydrocarbons/ leach from the tar or asphalt linings of these pipes ... resulting in increased concn of these cmpd in water reaching the consumers. ... Cement-lined pipes produce lower PAH concn, possibly because PAH's are adsorbed from water.
[National Research Council. Drinking Water & Health, Volume 4. Washington, DC: National Academy Press, 1981. 256]**PEER REVIEWED**

Analytic Laboratory Methods :

NIOSH Method 184: Polynuclear aromatic hydrocarbon (PAH) compounds are filtered from air with a glass fiber filter & collected on a silver membrane filter The samples collected on the membrane filter are extracted with benzene. They are separated on an alumina column with n-pentane-diethyl ether mixtures as eluting solvents. The collected fractions are analyzed by a Recording UV Spectrophotometer, Cary Model 14, Varian Associates, or equivalent. The method is sensitive & capable of measuring several (PAH) in a single filter. Any substance that hinders the chromatographic separation or that absorbs at the same wavelength as the sample compound may interfere. Benzo(a)pyrene is the 6th chem to elute among 9 specific PAHs. Its wavelength of maximum absorption is 382 NM. It has a baseline or background wavelength of 375, 390 NM. A normal sample size corresponds to 2 to 3 mg of benzene-soluble material. Precision is not determined
[U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual ofAnalytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1977-present.,p. V1 184-1]**PEER REVIEWED**

Analytic Laboratory Methods :

Contents of polycyclic aromatic hydrocarbons (PAHs) in the ointment and cosmetic bases liquid paraffin, yellow petrolatum, ichthammol and citric acid were determined by UV spectrophotometry (Japanese Pharmacopeia X), HPLC with fluorescence detection and TLC with fluorescence detection The UV method gave poor results due to its low sensitivity and selectivity, whereas the HPLC method gave a good selectivity and quant results. TLC had an ability to detect PAHs and may be used in the detn of the PAH limit. Benzo(a)pyrene, benzo(e) pyrene, benzo(k)fluoranthene, benzo(b)fluoranthene, benzo(g,h,i)perylene and dibenz(a,h)anthracene were nondetectable in liquid paraffin and citric acid, and benzo(a)pyrene was not detected in ichthammol. Yellow petrolatum contained 0.2-1 75 ng/g benzo(a)pyrene in 3 samples analyzed.
[Kawamura T, Nakagawa T; Iyakuhin Kenkyu 16 (2): 336-42 (1985)]**PEER REVIEWED**

Human Toxicity Excerpts :

Polycyclic aromatic compounds (PAC) are ubiquitous pollutants in urban air that may pose risks to human health In order to better assess the health risks associated with this class of compounds, a total of 67 polycyclic aromatic compounds that either have been identified (55) or are suspected to be present (12) in urban aerosol samples were tested for mutagenicity in a forward mutation assay based on human B-lymphoblastoid cells. The cell line used (designated hlAlv2) constitutively expresses the cytochrome p4501A1, which is known to be necessary for the metabolism of many promutagens. The polycyclic aromatic compounds tested included 39 polycyclic aromatic hydrocarbons (PAH), 19 oxygen-containing polycyclic aromatic hydrocarbons (oxy-PAH) and nine NO2-substituted polycyclic aromatic hydrocarbons (nitro-PAH) A total of 26 polycyclic aromatic hydrocarbons were mutagenic. In comparing the minimum mutagenic concentrations of the mutagenic polycyclic aromatic hydrocarbons with that of benzo(a)pyrene (B[a]P) it was found that dibenzo(a,l)pyrene (DB(al)P), cyclopenta(c,d))pyrene (CPP), naphtho(2,1-a)pyrene, dibenzo(a,e)pyrene (DB(ae)P) and l-methylbenzo(a)pyrene were 24 + or - 21, 6.9 + or - 4.2, 3.2 + or - 3.0, 2.9 + or - 2.9 and 1.6 + or - 1.4 times, respectively, more mutagenic than benzo(a)pyrene, and that dibenzo (a,k)fluoranthene and benzo(a)pyrene were approximately equally mutagenic. The 19 other mutagenic polycyclic aromatic hydrocarbons were between tested only phenalenone, 7H-benz(d,e)anthracen-7-one, 3-nitro-6H-dibenzo(b,d)pyran-6-one, cyclopenta(c,d)pyren-3(4H)-one, 6H-benzo(c,d)pyren-6-one (BPK) and anthanthrenequinone were mutagenic; however, with the exception of 6H-benzo(c,d)pyren-6-one, these were over 50 times less active than benzo(a)pyrene. 6H-benzo(c,d)pyren-6-one was benzo(a)pyrene. Seven of the nitro-polycyclic aromatic hydrocarbons were mutagenic including 9-nitroanthracene, l-nitrofluoranthene, 3-nitrofluoranthene, 1,3-dinitropyrene, 1,6-dinitropyrene (1,6-DNP) and 1,8-dinitropyrene. 1,6-dinitropyrene wasnic nitro-polycyclic aromatic hydrocarbons were between 20 and 380 times less active than benzo(a)pyrene. These results are discussed in terms of their relevance for determining the most important mutagens in ambient air. Based on reported concentrations of polycyclic aromatic compounds in ambient aerosols, it is possible that cyclopenta(c,d))pyrene, dibenzo(a,e)pyrene, dibenzo(a,l)pyrene and 6H-benzo(c,d)pyren-6-one could account for a greater proportion of the mutagenicity than benzo(a)pyrene in some aerosols
[Durant JL et al; Mutation Research 371 (3-4): 123-57 (1996)]**PEER REVIEWED**

Analytic Laboratory Methods :

A TLC/HPLC (HIGH PRESSURE LIQUID CHROMATOGRAPHY) PROCEDURE FOR DETERMINATION OF POLYCYCLIC AROMATIC HYDROCARBONS (PAH) OCCURRING IN ASPHALT FUMES (ADSORBED ON A PARTICULAR MATTER) IS DESCRIBED. THE METHOD IS BASED ON THE EXTRACTION OF ASPHALT FUME PARTICLES, COLLECTED ON GLASS-FIBER FILTERS, USING CARBON TETRACHLORIDE. A CLEAN UP STEP IS AIDED BY A TLC PROCEDURE ON ALUMINUM TRIOXIDE THINLAYER PLATES, USING A MIXTURE OF CYCLOHEXANE/ACETONE/ETHER AS THE MOBILE PHASE. UNDER UV-LIGHT, THE PAH ARE INDICATED AS FLUORESCENT SPOTS. SEPARATION OF THE COLLECTED PAH INTO INDIVIDUAL COMPONENTS & THEIR IDENTIFICATION IS PERFORMED BY THE AID OF A HPLC PROCEDURE.
[RIETZ EB; ANAL LETT 12 (12): 143-54 (1979)]**PEER REVIEWED**

Cleanup Methods :

.. In surface waters, one-third of the total PAH is bound to larger suspended particles, a third is bound to finely dispersed particles, and the last third is present in dissolved form The particle-bound portion of polycyclic aromatic hydrocarbons (PAH) can be removed by sedimentation, flocculation, and filtration processes. The remaining one-third dissolved PAH usually requires oxidation for partial removal/transformation. /polynuclear aromatic hydrocarbons/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons (Draft) p.C-4 (1980)]**PEER REVIEWED**

Cleanup Methods :

This method incorporates procedures for the destruction of laboratory wastes contaminated with PAH using an aqueous saturated potassium permanganate solution. This method has been tested for wastes contaminated with the following PAH Benz(a)anthracene, 7,12-dimethylbenz(a)anthracene, benzo(a)pyrene, 3-methylcholanthrene, and 7-bromomethylbenz(a)anthracene. It has been studied collaboratively with a solution of benz(a)anthracene + benzo(a)pyrene + 7,12-dimenthylbenz(a)anthracene and a solution of 3-methylcholanthrene + dibenz(a,h)anthracene in cyclohexane. The method affords better than 95% destruction of all of the PAH tested except for dibenzo(a,h)anthracene, for which only variable and incomplete destruction could be achieved
[Castegnaro, M., G. Grimmer, O. Hutzinger, W. Karcher, H. Kunte, M. LaFontaine, E.B. Sansone, G. Telling, and S.P. Tucker (eds.). Laboratory Decontaminationand Destruction of Carcinogens in Laboratory Wastes:

Some Polycyclic Aromatic Hydrocarbons. IARC Publications No. 49. Lyon, France: International Agency for Research on Cancer, 1983. 31]**PEER REVIEWED**

Human Toxicity Excerpts :

Previous studies in this laboratory have shown that polycyclic aromatic hydrocarbons (PAHs) alter Ca(2+) homeostasis and inhibit activation of both B and T lymphocytes obtained from rodents and humans. In the present studies, we demonstrate that a-naphthoflavone (ANF), an inhibitor of cytochrome p4501A activity, reduced the Ca(2+) elevation produced by benzo(a)pyrene in human peripheral blood mononuclear cell (HPBMC) lymphocytes. These results suggested that benzo(a)pyrene metabolites may play a role in intracellular Ca(2+) homeostasis in human lymphocytes. Reactive oxidative intermediates of benzo(a)pyrene produced in human peripheral blood mononuclear cell are known to be highly carcinogenic and have also been shown to be immunosuppressive We examined the effects of benzo(a)pyrene (BaP), 7,12-dimethylbenz(a)anthracene (DMBA), benzo(c)pyrene (BeP), and anthracene, as well as certain benzo(a)pyrene metabolites, on the levels of intracellular Ca(2+) and glutathione in human peripheral blood mononuclear cell. While benzo(a)pyrene, 7,12-dimethylbenz (a)anthracene, benzo(e)pyrene, and anthracene did not cause a statistically significant decrease in GSH in human peripheral blood mononuclear cell at concentrations of 1 or 10 uM following a 6-, 48-, or 72-hr exposure, reactive benzo(a)pyrene metabolites including 4,5-epoxide benzo(a)pyrene and 7,8-diol-9,10-epoxide benzo(a)pyrene consistently produced a 20-30% depletion of glutathione in human peripheral blood mononuclear cell following a 6-hr treatment period. These benzo(a)pyrene metabolites also elevated intracellular Ca(2+) in human peripheral blood mononuclear cell during a 6-hr incubation. Results of these experiments suggest that metabolism of benzo(a) pyrene to certain epoxide metabolites lay be responsible for sulfhydryl damage leading to transient GSH depletion and Ca(2+) elevation. These results are consistent with the hypothesis that sulfhydryl damage by certain PAH metabolites may lead to altered Ca(2+) homeostasis, leading to inhibition of cell activation and proliferation in human peripheral blood mononuclear cell
[Romero DL et al; Toxicol and Applied Pharmacol 144 (1): 62-9 (1997)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Buffalo river sediment extracts contained polynuclear aromatic hydrocarbons (PAH) which caused skin darkening, hyperplasia, skin papillomas, mild coarsening and local pigmentations in the brown bullhead (Ictalurus nebulosus). Sixteen PAH were identified in the sediment extract: fluorene, phenanthrene, anthracene, fluoranthene, 2-methylphenanthrene, pyrene, 2-methylanthracene, benzanthracene, chrysene, perylene, benzo(f)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, benzo(g,h,i)perylene, and indeno (1,2,3-c,d)pyrene
[Black JJ; Polynucl Aromat Hydrocarbons Int Symp 7th 99-11 (1983)]**PEER REVIEWED**

Human Toxicity Excerpts :

The relative contributions of biologic and environmental factors on embryo-fetal development were elucidated in a population of pregnant women who were exposed to varying amounts of active cigarette smoke and women who were not exposed to cigarette smoke. The neonatal weight at birth, placental weight at delivery, duration of pregnancy, and placental xenobiotic (polynuclear aromatic hydrocarbon, PAH) metabolism potential were assessed in this population. The overall metabolic capability in exposed and unexposed placental tissue was measured by in vitro assays using microsomes and a polynuclear aromatic hydrocarbon substrate, benzo[a]pyrene (B[a]P) Toxicity potential was determined by benzo[a]pyrene-metabolite-DNA adduct generation under the same incubation condition. Cigarette smoke exposure increased the overall polynuclear aromatic hydrocarbon metabolism potential in placental tissues by approximately 200% (nonsmoker 176.2 + or - 33.6, n = 25; smoker 524.5 + or - 75.5, n = 32 pmol/mg protein) whereas polynuclear aromatic hydrocarbon-DNA adduct formation potential did not increase significantly over the basal level (nonsmoker 5002 + or - 830, n = 15, smoker 6172 + or - 1443, n = 22 fmol benzo[a]pyrene equivalent/umol DNA/mg protein). Exposure to cigarette smoke during pregnancy is deleterious to fetal development as reflected by reduced neonatal weight at birth. In contrast, placental weight reduction is indistinct, but placentae expressed markedly augmented overall xenobiotic (PAH) metabolism capability in response to cigarette smoke exposure during pregnancy, indicating placental metabolism may be an important mediator of adverse effects induced by such xenobiotic exposure.
[Sanyal MK et al; Reprod Toxicol 8 (5): 411-8 (1994)]**PEER REVIEWED**

Disposal Methods :

The method incorporates procedures for the destruction of laboratory wastes contaminated with PAH using concentrated sulfuric acid The method has been tested for wastes contaminated with the following PAH: Benz(a)anthracene, 7,12-dimethylbenz(a)anthracene, benzo(a) pyrene, 3-methylcholanthrene, 7-bromomethylbenz(a)anthracene, and dibenz(a,h)anthracene. It has been studied collaboratively using solutions of benz(a)anthracene + benzo(a)pyrene + 7,12-dimethylbenz(a)anthracene and solutions of methylcholanthrene + dibenz(a,h) anthracene in DMF and DMSO. ... The method affords better than 99% destruction in all solutions tested.
[Castegnaro, M., G. Grimmer, O. Hutzinger, W. Karcher, H. Kunte, M. LaFontaine, E.B. Sansone, G. Telling, and S.P. Tucker (eds.). Laboratory Decontaminationand Destruction of Carcinogens in Laboratory Wastes: Some Polycyclic Aromatic Hydrocarbons. IARC Publications No. 49. Lyon, France: International Agency for Research on Cancer, 1983. 25]**PEER REVIEWED**

Analytic Laboratory Methods :

A 4-STEP METHOD FOR THE REPRODUCIBLE ANALYSIS OF POLYNUCLEAR AROMATIC HYDROCARBONS (PAH) IN SMALL QUANTITIES OF CIGARETTE SMOKE CONDENSATE (CSC) IS PRESENTED. PAH WERE ISOLATED FROM AS

LITTLE AS 1 G OF CSC BY SOLVENT PARTITION, COLUMN CHROMATOGRAPHY, & ANALYSIS GEL FILTRATION (GF). THE GF ISOLATE WAS ANALYZED BY GAS CHROMATOGRAPHY. /POLYNUCLEAR AROMATIC HYDROCARBONS/
[SEVERSON RF ET AL; ANAL CHEM 48 (13): 1866 (1976)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Several well-documented examples of human exposure to carcinogens involve complex mixtures of polycyclic aromatic hydrocarbons (PAHs). Although the biological properties of many pure polycyclic aromatic hydrocarbons have been investigated, less is known about their effects when present as components of mixtures. As the ability to form DNA adducts in vivo is generally indicative of carcinogenic activity of polycyclic aromatic hydrocarbons, we have compared the DNA binding potencies of dibenzo(a,e)pyrene (DB(a,e)P), dibenzo (a,h)pyrene (DB(a,h)P), dibenzo(a,i)pyrene (DB(a,i)P), dibenzo(a,l)pyrene (DB(a,l)P) and benzo(a)pyrene (B(a)P), when applied topically, either singly or in combination, to the skin of male Parkes mice DNA isolated from the skin and lungs was analyzed by 32P-postlabelling. The adducts formed by each polycyclic aromatic hydrocarbon exhibited markedly different chromatographic mobilities on polyethyleneimine-cellulose TLC plates. The relative binding potencies of the compounds in both skin and lungs were: dibenzo(a,l)pyrene > dibenzo(a,i)pyrene > dibenzo(a,e)pyrene, in good agreement with their reported carcinogenicities in mouse skin. The majority of adducts were removed from DNA within 21 days of treatment, but low levels of adducts were found to persist for at least 3 months in both tissues. When dibenzo(a,l)pyrene, dibenzo(a,e)pyrene and benzo(a)pyrene were applied together to mouse skin, a total binding 31% lower than expected was detected, while with a mixture of dibenzo(a,e)pyrene and benzo(a)pyrene the binding to DNA in skin was 65% higher than expected from the binding levels of the carcinogenes when applied singly. Other binary combinations of these three polycyclic aromatic hydrocarbons gave adduct levels similar to the sum of the binding levels of the individual components when applied singly. The results demonstrate the usefulness of 32P-post-labelling for the assessment of the DNA binding potencies of polycyclic aromatic hydrocarbons in mouse tissues, and for the detection of interactions between components of mixtures of carcinogens.
[Hughes NC, Phillips DH; Carcinogenesis (EYNSHAM); 11 (9): 1611-20 (1990)]**PEER REVIEWED**

Interactions :

Iron oxides are present in many occupational atmospheres mainly in iron ore mines and in steel industry. Among these workers, epidemiological studies indicated an excess of lung cancer deaths. In mines, it was difficult to involve iron oxides exposure because there are other possible causes as radon, polycyclic aromatic hydrocarbon (PAH) present in diesel exhausts, silicosis or siderosis. The contradictory results of these studies are due to the differences of exposure levels or to the presence or not of these cofactors or of a sufficient prevention. But generally the results agree with an interaction of iron oxide dusts and smoking habits. It is unclear if this interaction supports an additive or multiplicative risk of lung cancer. Experimental studies with Fe203 showed that these particles are able to induce lung cancers only in the presence of polycyclic aromatic hydrocarbon when administered to animals. In vitro studies permitted to observe an interaction in the metabolism of benzo(a)pyrene (BaP) leading to a higher level of precursors of the ultimate carcinogen. As this metabolism of benzo(a)pyrene is known to be enhanced during lipoperoxidation, it is possible to involve this mechanism with Fe203. After phagocytosis and dissolution with production of ferric ions, Fe203 can enhance the production of reactive oxygen species responsible of damaging 60th lipidic constituents and DNA. Fe304 and mainly FeO may be more toxic, introducing directly ferrous ions in the cells after dissolution, but the canerogenicity of these compounds is unknown, making necessary to develop research
[Haguenoer JM et al; Cent Eur J Public Health (4): 41-5 (1996)]**PEER REVIEWED**

Analytic Laboratory Methods :

OSW Method 8275A. Semivolatile Organic Compounds (PAHs and PCBs) in Soils/Sludges and Solid Wastes Using Thermal Extraction/Gas Chromatography/Mass Spectrometry (TE/GC/MS), soil/waste, TEGCMS.
[USEPA; EMMI. Environmental Monitoring Methods Index. Version 2.0 NTIS PB-95-502415 (1995)]**PEER REVIEWED**

Human Toxicity Excerpts :

Exposures to other chemical mixtures that contain PAHs, such as cigarette smoke, coal tar, coal tar pitch, and bitumens, have been associated with increased incidences of lung cancer in humans. /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Human Toxicity Excerpts :

Workers exposed to creosote containing numerous PAHs developed skin tumors .. /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Human Toxicity Excerpts :

Mortality studies have demonstrated that exposure to coke oven emissions, which contain a variety of PAHs, caused increased incidences of lung and genitourinary cancer mortality in coke oven workers .. . /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Interactions :

Benzo[a]pyrene (B[a]P) is able to inhibit the mutagenicity of 1-nitropyrene (1-NP) through the reduction of nitroreductase activity and formation of adducts with DNA. The relationships between the chemical structure of 9 polycyclic aromatic hydrocarbons (PAHs) and antagonistic effects on the 1-nitropyrene-induced mutation were evaluated by the binary mixtures of 1-nitropyrene and polycyclic aromatic hydrocarbons with Salmonella typhimurium TA98 in the absence of S9 mix. Remarkably different antagonistic effects of 9 polycyclic aromatic hydrocarbons on the mutagenicity of 1-nitropyrene were observed. Among the tested polycyclic aromatic hydrocarbons, coronene demonstrates the most antagonistic potential followed by benzo[g,h,i]perylene (B[g,h,i]P), benzo[e]pyrene (B[e]P), dibenzo[a,h]pyrene (DB[a,h]P), benzo[a]pyrene (B[a]P) and pyrene. Naphthalene, anthracene, and chrysene had only minor inhibitory activity on the 1-nitropyrene mutagenicity The modifying effects of polycyclic aromatic hydrocarbons on the nitroreductase activity of TA98 strains in the presence of 1-nitropyrene were further examined from the production of 1-AP. The statistical analytical data showed that the inhibitory effect of polycyclic aromatic hydrocarbons on the mutagenicity of 1-nitropyrene significantly correlated with their effects on the nitroreductase activity ($r = -0.69$, $p < 0.05$) In addition, the formation of 1-nitropyrene-DNA adducts of the binary mixtures of 1-nitropyrene and polycyclic aromatic hydrocarbon was determined by the 32P-postlabeling method. The results indicated that the modulatory effects of polycyclic aromatic hydrocarbons on the formation of 1-nitropyrene-DNA adducts were correlated well with their antagonistic activity ($r = -0.91$, $P < 0.011$. From the above results, the relationships between the chemical structure of polycyclic aromatic hydrocarbons and the antagonistic effects on the 1-nitropyrene mutagenicity were revealed by the surface area and electronic parameters of polycyclic aromatic hydrocarbons. The planar molecular area of polycyclic aromatic hydrocarbons was more convincingly correlated with the antagonistic effect on the mutagenicity of 1-nitropyrene ($r = -0.81$, $p < 0.01$) than that with the difference in energy, delta E, between EHOMO and ELUMO ($r = 0.69$, $p < 0.05$). According to the above, two possible mechanisms are involved in the interactive effect of the binary mixtures: (1) a higher binding affinity with nitroreductase for polycyclic aromatic hydrocarbons having a large planar surface area; and (2) a high energy of interaction between 1-nitropyrene and polycyclic aromatic hydrocarbons with a low delta E might decrease the nitroreductive capability
[Cherng SH et al; H Mutat Res 367 (4): 177-85 (1996)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

Rats and mice were exposed to combustion gases of coal-burning furnace enriched with benzo(a)pyrene (50-90 ug/cu m) and other polycyclic aromatic hydrocarbons (PAH) 16 hr/day, 5 days/wk. After approx 22-mo exposure, the incidence of lung neoplasm was approx 10-fold above controls.
[Heinrich U et al; Exp Pathol 29 (1): 29-34 (1986)]**PEER REVIEWED**

Interactions :

Several well-documented examples of human exposure to carcinogens involve complex mixtures of polycyclic aromatic hydrocarbons (PAHs). Although the biological properties of many pure polycyclic aromatic hydrocarbons have been investigated, less is known about their effects when present as components of mixtures. As the ability to form DNA adducts in vivo is generally indicative of carcinogenic activity of polycyclic aromatic hydrocarbons, we have compared the DNA binding potencies of dibenzo(a,e)pyrene (DB(a,e)P), dibenzo(a,h)pyrene (DB(a,h)P), dibenzo(a,i)pyrene (DB(a,i)P), dibenzo(a,l)pyrene (DB(a,l)P) and benzo(a)pyrene (B(a)P), when applied topically, either singly or in combination, to the skin of male Parkes mice DNA isolated from the skin and lungs was analyzed by 32P-postlabelling. The adducts formed by each polycyclic aromatic hydrocarbon exhibited markedly different chromatographic mobilities on polyethyleneimine-cellulose TLC plates. The relative binding potencies of the compounds in both skin and lungs were: dibenzo(a,l)pyrene > dibenzo(a,i)pyrene > dibenzo(a,e)pyrene, in good agreement with their reported carcinogenicities in mouse skin. The majority of adducts were removed from DNA within 21 days of treatment, but low levels of adducts were found to persist for at least 3 months in both tissues. When dibenzo(a,l)pyrene, dibenzo(a,e)pyrene and benzo(a)pyrene were applied together to mouse skin, a total binding 31% lower than expected was detected, while with a mixture of dibenzo(a,e)pyrene and benzo(a)pyrene the binding to DNA in skin was 65% higher than expected from the binding levels of the carcinogenes when applied singly. Other binary combinations of these three polycyclic aromatic hydrocarbons gave adduct levels similar to the sum of the binding levels of the individual components when applied singly. The results demonstrate the usefulness of 32P-post-labelling for the assessment of the DNA binding potencies of polycyclic aromatic hydrocarbons in mouse tissues, and for the detection of interactions between components of mixtures of carcinogens.
[Hughes NC, Phillips DH; Carcinogenesis (EYNSHAM); 11 (9): 1611-20 (1990)]**PEER REVIEWED**

Human Toxicity Excerpts :

Previous studies have shown that polycyclic aromatic hydrocarbons (PAHs) mobilize intracellular Ca2+ in human T cells by inositol trisphosphate-dependent mechanisms resulting from activation of phospholipase C-gamma by SRC-related protein tyrosine kinases, thereby mimicking antigen-receptor activation. Ca2+ appears to play an important second messenger role in growth factor control of cell proliferation in human mammary epithelial cells (HMEC) such as the epidermal growth factor receptor pathway. The purpose of the present studies was to determine if polycyclic aromatic hydrocarbons are able to increase intracellular Ca2+ in primary cultures of human mammary epithelial cells and increase cell proliferation Two carcinogenic and two non-carcinogenic polycyclic aromatic hydrocarbons were tested for their ability to increase intracellular Ca2+ in human mammary epithelial cells. The carcinogenic polycyclic aromatic hydrocarbons dimethylbenz(a)anthracene (DMBA) and benzo[a] pyrene (BaP) were able to cause Ca2+ elevation in human mammary epithelial cells at early time points (2 hr) and caused sustained alterations in Ca2+ homeostasis (18 hr). Dimethylbenz(a)anthracene showed maximal effects at early time points (2 hr), while benzo(a)pyrene showed maximal effects on sustained Ca2- (18 hr). 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), a potent dioxin and tumor promoter, produced maximal Ca2+ elevation at 2 hr, with a return to near

baseline levels by 6 hr. The non-carcinogenic polycyclic aromatic hydrocarbons benzo[e]pyrene and anthracene did not significantly alter intracellular Ca2+ at any time point. alpha-Naphthoflavone significantly reduced the Ca2+ response induced by benzo(a)pyrene treatment, but not by dimethylbenz(a)anthracene or 2,3,7,8-Tetrachlorodibenzo-p-dioxin, suggesting that p450 lA or lB metabolism of benzo(a)pyrene may be important in the sustained Ca2+ elevating response. In evaluating the effects of benzo(a)pyrene on human mammary epithelial cells proliferation, benzo(a)pyrene was found to increase the number of cells recovered after 4 days in culture in the absence or presence of various concentrations of epidermal growth factor. These studies provide initial evidence that Ca2+ signaling may be associated with mitogenesis in human mammary epithelial cells, which may play a role in tumor promotion and progression produced by polycyclic aromatic hydrocarbons.
[Tannheimer SL et al; Carcinogenesis 18 (6): 1177-82 (1997)]**PEER REVIEWED**

Human Toxicity Excerpts :

Dermal exposure to coal tar and shale oils containing PAHs have been associated with increased incidences of skin tumors in humans .. . /Polycyclic aromatic hydrocarbons/
[DHHS/NIEHS; Seventh Annual Rpt on Carcinogens Summary p. 330 (1994)]**PEER REVIEWED**

Analytic Laboratory Methods :

The X-ray excited optical luminescence (XEOL) of a concentrate in n-heptane of the neutral fraction isolated from by-products of coal combustion and conversion, and from shale and fuel oils was utilized to obtain profiles of their PAH content. Components which should be unequivocally identified include benzo(a)anthracene, BaP, benzo(e)pyrene, benzo(ghi)perylene, pyrene, benzo(k)fluoranthene, and coronene.
[Fassel VA et al; Analytical Chem 52 (1): 159-64 (1980)]**PEER REVIEWED**

Analytic Laboratory Methods :

ULTRASONIC EXTRACTION OF AIRBORNE PARTICULATE MATERIAL ON HI-VOL FILTERS IS DESCRIBED. ALMOST ALL POLAR COMPOUNDS ARE REMOVED DURING EXTRACTION BY ADSORPTION ON THE SURFACE OF SHREDDED GLASS FIBERS & CONTROLLED PORE GLASS POWDER (CPG). NON-POLAR POLYNUCLEAR AROMATIC HYDROCARBONS (PAH) IN THE EXTRACTS ARE SEPARATED AT ROOM TEMP BY HIGH PRESSURE LIQUID CHROMATOGRAPHY (HPLC) ON REVERSE PHASE VYDAC USING ACETONITRILE.WATER (70:30 VOL/VOL) AS CHROMATOGRAPHIC SOLVENT B(A)P ELUTES IN APPROX 14 MIN. PRECISION & ACCURACY MEASUREMENTS INDICATE FULL RECOVERY & GOOD EXTRACTION REPRODUCIBILITY. DETECTION LIMIT FOR B(A)P AT F 290/389 IS LESS THAN 100 PG. TOTAL ANALYSIS TIME IS APPROX 1.5 HR.
[GOLDEN C, SAWICKI E; ANAL LETT 11 (12): 1051-62 (1978)]**PEER REVIEWED**

Clean Water Act Requirements :

For the maximum protection of human health from the potential carcinogenic effects due to exposure of polynuclear aromatic hydrocarbons through ingestion of contaminated water and contaminated aquatic organisms, .. therefore, the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 1x10-5, 1x10-6, and 1x10-7. The corresponding criteria /for ambient water/ are 28 0 ng/l, 2.8 ng/l, and 0.28 ng/l, rspectively. If the above estimates are made for consumption of aquatic organisms only, excluding consumption of water, the levels are 344 0 ng/l, a 31.1 ng/l, and 3.11 ng/l respectively. /Polynuclear aromatic hydrocarbons based on benzo (a)pyrene as the model PAH/
[USEPA; Ambient Water Quality Criteria Doc: Polynuclear Aromatic Hydrocarbons p.C-121 (1980)] **QC REVIEWED**

Non-Human Toxicity Excerpts :

In animal studies, exposure to high levels of diesel exhaust particulates overwhelms the normal clearance mechanisms and results in lung burdens of diesel exhaust particulates that exceed those predicted from observations at lower exposure concentrations. A variable amount of the mass of diesel exhaust particulates is extractable with strong organic solvents. The extracted material contains more than a thousand individual compounds and is mutagenic in a number of bacterial and mammalian cell assays. Bioassay-directed chemical analysis of diesel exhaust particulates had identified several hundred compounds. Many are PAHs, some of which are considered to have human carcinogenic potential. The association of benzo(a)pyrene and nitropyrene with diesel exhaust particulates prolongs their retention in the lungs
[McClellan RD; Annu Rev Pharmacol Toxicol 27: 279-300 (1987)]**PEER REVIEWED**

Emergency Medical Treatment :

EMT Copyright Disclaimer:

Portions of the POISINDEX(R) database are provided here for general reference. THE COMPLETE POISINDEX(R) DATABASE, AVAILABLE FROM MICROMEDEX, SHOULD BE CONSULTED FOR ASSISTANCE IN THE DIAGNOSIS OR TREATMENT OF

The following Overview, *** POLYNUCLEAR AROMATIC HYDROCARBONS.***, is relevant for this HSDB record chemical.

Life Support:

- This overview assumes that basic life support measures have been instituted.

Clinical Effects:

SUMMARY OF EXPOSURE

0.2.1.1 ACUTE EXPOSURE

- In general, **PAHs** have a low order of acute toxicity in humans.
- **PAHs** and other compounds found in COAL TAR can produce a variety of non-cancer effects with chronic exposure. Chronic effects include:
  1. EYES - Photosensitivity and irritation.
  2. RESPIRATORY - Irritation with cough and bronchitis.
  3. MOUTH - Leukoplakia.
  4. DERMAL - "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, dermal burns, photosensitivity, acneiform lesions, irritation.
  5. HEPATIC/RENAL - Mild hepatotoxicity or mild nephrotoxicity (animals).
  6. GENITOURINARY - Hematuria.
- CANCER is the most significant **PAH** toxicity endpoint.
  1. Increased incidences of cancers of the skin, bladder, lung and gastrointestinal tract have been described in **PAH**-exposed workers.

RESPIRATORY

0.2.6.1 ACUTE EXPOSURE

- Irritation, chronic cough, bronchitis, and bronchogenic cancer can occur with chronic exposure.

GASTROINTESTINAL

0.2.8.1 ACUTE EXPOSURE

- Leukoplakia and cancers of the lip and oral cavity can develop with chronic exposure.

HEPATIC

0.2.9.1 ACUTE EXPOSURE

- Mild hepatotoxicity has been reported in **PAH**-exposed rats.

GENITOURINARY

0.2.10.1 ACUTE EXPOSURE

- Hematuria, kidney and bladder cancer are possible effects of chronic exposure. Mild nephrotoxicity has been documented in **PAH**-exposed rats.

HEMATOLOGIC

0.2.13.1 ACUTE EXPOSURE

- Agranulocytosis, anemia, leukopenia, and pancytopenia developed in rats chronically fed **PAHs**.

DERMATOLOGIC

0.2.14.1 ACUTE EXPOSURE

- PRECANCEROUS LESIONS - "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, dermal burns, acneiform lesions, photosensitization and cancer may develop following chronic exposure.

IMMUNOLOGIC

0.2.19.1 ACUTE EXPOSURE

- An effect of **PAHs** on immune function might aid in the development of neoplasms. A number of **PAH** compounds are immunotoxic, and some suppress selective components of the immune system.

REPRODUCTIVE HAZARDS

- In experimental animal studies, **PAHs** and metabolites cross the placenta. Female offspring of experimental animals exposed to **PAHs** during pregnancy have a decrease in the number of functional oocytes, sometimes such that they are infertile.
- **PAHs** are lipophilic and are excreted in breast milk, allowing for secondary exposure of nursing infants, although the potential significance of such exposure has not been determined.

CARCINOGENICITY

0.2.21.1 IARC CATEGORY

- PNAs AS A GROUP -
  1. Varies. Classified as Group 2A - probably carcinogenic to humans, to Group 3 - not classifiable as to its carcinogenicity to humans (IARC, 1989).
- INDIVIDUAL PNAs -
  1. ANTHRACENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  2. BENZ[A]ANTHRACENE - 2A, probably carcinogenic to humans (IARC, 1987).
  3. BENZO[K]FLUORANTHENE - 2B, possibly carcinogenic to humans (IARC, 1987).
  4. BENZO[GHI]PERYLENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  5. BENZO[A]PYRENE - 2A - probably carcinogenic to humans (IARC, 1987).
  6. BENZO[E]PYRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  7. CHRYSENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  8. CORONENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  9. DIBENZ[A,H]ACRIDINE - 2B - possibly carcinogenic to humans (IARC, 1987).
  10. DIBENZ[A,H]ANTHRACENE - 2A - probably carcinogenic to humans (IARC, 1987).
  11. 7H-DIBENZO[C,G]CARBAZOLE - 2B - possibly carcinogenic to humans (IARC, 1987).
  12. PHENANTHRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
  13. PYRENE - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1987).
- RELATED SUBSTANCES & PROCESSES -
  1. COAL GASIFICATION; COAL-TAR PITCHES; COAL-TARS - 1 - carcinogenic to humans (IARC, 1987).
  2. COKE PRODUCTION - 1 - carcinogenic to humans (IARC, 1987).
  3. CREOSOTES - 2A - probably carcinogenic to humans (IARC, 1987).
  4. UNTREATED AND MILDLY TREATED MINERAL OILS - 1 - carcinogenic to humans (IARC, 1989).
  5. HIGHLY REFINED MINERAL OILS - 3 - not classifiable as to its carcinogenicity to humans (IARC, 1989).

0.2.21.2 HUMAN OVERVIEW

- Cancer is the most significant **PAH** toxicity endpoint. Some, but not all, **PAHs** are carcinogens. Certain **PAH** parent compounds are weak carcinogens, only becoming potent carcinogens after undergoing metabolism. Chronic or repeated exposure increases the likelihood of cancer initiation, as well as the potential for metabolism of a **PAH** procarcinogen to a carcinogen.
- Increased incidences of skin, bladder, lung, and possibly gastrointestinal tract cancers have been described in **PAH**-exposed workers, particularly associated with coal carbonization, coal gasification, and coke oven work.

0.2.21.3 ANIMAL OVERVIEW

- Lung tumors were induced in female mice from inhalation of exhausts containing **PAHs** (Schulte et al, 1994).

GENOTOXICITY

- CYTOGENETIC MARKERS OF HUMAN **PAH** EXPOSURE - Cells containing high frequencies of chromosomal aberrations were a sensitive marker for exposure to **PAHs** in coke oven and graphic electrode plant workers, compared with unexposed controls. There was no relation between cytogenetic findings and levels of benzo(a)pyrene-hemoglobin adducts (Buchet et al, 1995).

1. Arylamines derived from carcinogenic **PAHs** are mutagenically activated through S9-mediated metabolism of the related amine (Fu et al, 1982).
2. Nitro-**PAH** derivatives are potent bacterial mutagens. The mutagenic activity is dependent on enzymatic reduction of the nitro group and may require oxidative

metabolism (Grosovsky et al, 1999; Rosenkranz & Mermelstein, 1985).
3. In cultured human cells, benzo(a)pyrene and 7,12-demethylbenz(a)anthracene only caused a significant increase in T6 guanine-resistant mutations in the presence of cultured rat hepatocytes (Tong et al, 1981).
4. Nitrated **PAHs** have caused dose-dependent cell transformations in Syrian hamster embryo cells (DiPaolo et al, 1983). Metabolic reduction of the nitro- group on **PAHs** may be involved in their mutagenic effects.
5. Persons with a high degree of inducibility of the enzyme, aryl hydrocarbon hydroxylase, may be a high-risk population (ATSDR, 1993).

o MUTAGENIC INTERMEDIATES -
1. The **PAH** metabolic intermediates, diol-epoxides, are presumably mutagenic and can react to form DNA adducts, which may affect normal cellular replication (ATSDR, 1993; Ellenhorn & Barceloux, 1988; Pike, 1992; Amin et al, 1993; Ronai et al, 1994).

o LACK OF EFFECT
1. Amongst a small group of Swedish road pavers exposed to asphalt fumes, there were no significant increases in sister chromatic exhanges or micronuclei in peripheral blood/lymphocytes as compared to unexposed controls (Jarvholm et al, 1999).

Laboratory:

- **PAHs** have been determined in the blood and tissues of experimental animals. Direct biologic measurement of **PAHs** currently is not clinically useful or cost-effective. Indirect methods of determining exposure are available but have not yet proven clinically useful (ATSDR, 1990).
- Acute respiratory effects in persons at **PAH**-containing workplaces are typically due to other toxic agents at the worksite (ATSDR, 1990). Arterial blood gases, chest x-ray, and other monitoring may be indicated, based on the patient's presentation and the exposure characteristics.
- Chronic effects, particularly cancer, are more common than acute toxicity. Routine monitoring and physical assessments (e.g, complete blood count, hepatic and renal function tests, chest xray and pulmonary function tests, dermal assessments) of individuals with significant exposure is recommended, even in the absence of symptoms (ATSDR, 1990).

Treatment Overview:

ORAL EXPOSURE
- Toxicity from these substances involves chronic exposure, toxicity after acute ingestions is unlikely and gastric decontamination is generally NOT indicated.

INHALATION EXPOSURE
- DECONTAMINATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer 100 percent humidified supplemental oxygen with assisted ventilation as required.
- Inhalational exposure to **PAHs** may be complicated by exposure to other substances which produce acute respiratory and systemic effects. Treat according to clinical presentation and exposure history.
1. If bronchospasm and wheezing occur, consider treatment with inhaled sympathomimetic agents.
2. Carefully observe patients with inhalation exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.
3. Monitor arterial blood gases, pulmonary function, and chest x-ray for patients with significant exposure.

EYE EXPOSURE
- DECONTAMINATION: Exposed eyes should be irrigated with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.

DERMAL EXPOSURE

- DECONTAMINATION: Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists.
- Treat dermal irritation or burns with standard topical therapy. Patients developing dermal hypersensitivity reactions may require treatment with systemic or topical corticosteroids or antihistamines.
- Some chemicals can produce systemic poisoning by absorption through intact skin. Carefully observe patients with dermal exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.

Range of Toxicity:

- The minimum lethal human dose to this agent has not been delineated.
- The maximum tolerated human exposure to this agent has not been delineated.

[Rumack BH: POISINDEX(R) Information System. Micromedex, Inc., Englewood, CO, 2001; CCIS Volume 109, edition exp August, 2001. Hall AH & Rumack BH (Eds):TOMES(R) Information System. Micromedex, Inc., Englewood, CO, 2001; CCIS Volume 109, edition exp August, 2001.] **PEER REVIEWED**

HYDROGEN CYANIDE
Synonym: **cyanide**
CASRN: 74-90-8
For other data, click on the Table of Contents

Best Sections

Environmental Fate/Exposure Summary :

Hydrogen cyanide's production and use as a starting material in the manufacture of acrylates, cyanide salts, herbicides and dyes as well as its former use as a fumigant resulted in its release to the environment through various waste streams. If released to air, a vapor pressure of 742 mm Hg at 25 deg C indicates hydrogen cyanide will exist solely as a vapor in the ambient atmosphere. Vapor-phase hydrogen cyanide will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals, the half-life for this reaction in air is estimated to be 535 days. If released to soil, hydrogen cyanide is expected to have very high mobility. Volatilization from moist soil surfaces is expected to be an important fate process based upon a Henry's Law constant of 1 33X10-4 atm-cu m/mole. Hydrogen cyanide may volatilize from dry soil surfaces based upon its vapor pressure. Hydrogen cyanide can be biodegraded by acclimated microbial cultures and sludges, but is usually toxic at high concentrations to unacclimated microbial systems. If released into water, hydrogen cyanide is not expected to adsorb to suspended solids and sediment in water. Volatilization from water surfaces is expected to be an important fate process based upon this compound's estimated Henry's Law constant. Estimated volatilization half-lives for a model river and model lake are 3 hours and 3 days, respectively. A pKa of 9.2 indicates that the dissociated form may exist at high pH. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Occupational exposure to hydrogen cyanide may occur through inhalation and dermal contact with this compound at workplaces where hydrogen cyanide is produced or used. The general population may be exposed to hydrogen cyanide from automobile exhaust and waste incinerators. (SRC)
PEER REVIEWED

Fire Fighting Procedures :

Evacuation: If fire becomes uncontrollable or container is exposed to direct flame - consider evacuation of one-half (1/2) mile radius. /**Hydrogen cyanide**, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Fire Fighting Procedures :

If material on fire or involved in fire: Do not extinguish fire unless flow can be stopped. Use water in flooding quantities as fog Use "alcohol" foam, dry chemical or carbon dioxide. Cool all affected containers with flooding quantities of water. Apply water from as far a distance as possible. Solid streams of water may be ineffective. /**Hydrogen cyanide**, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Preventive Measures :

If material not on fire and not involved in fire: Keep sparks, flames, and other sources of ignition away. Keep material out of water sources and sewers. Build dikes to contain flow as necessary Attempt to stop leak if without undue personnel hazard Use water spray to knock-down vapors. /**Hydrogen cyanide**, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Preventive Measures :

Personnel protection: Avoid breathing vapors. Keep upwind. ... Avoid bodily contact with the material. .. Do not handle broken packages unless wearing appropriate personal protective equipment. Wash away any material which may have contacted the body with copious amounts of water or soap and water. /**Hydrogen cyanide**, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Cleanup Methods :

Environmental considerations - Land spill: Dig a pit, pond, lagoon, holding area to contain liquid or solid material. /SRP: If time permits, pits, ponds, lagoons, soak holes, or holding areas should be sealed with an impermeable flexible membrane liner./ Dike surface flow using soil, sand bags, foamed polyurethane, or foamed concrete. Absorb bulk liq with fly ash or cement powder. /Hydrogen cyanide, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Cleanup Methods :

Environmental considerations - Water spill: Use natural barriers or oil spill control booms to limit spill travel. Neutralize with agricultural lime (CaO), crushed limestone (CaCO2), or sodium bicarbonate (NaHCO3). /Hydrogen cyanide, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized, hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Cleanup Methods :

Environmental considerations - Air spill: Apply water spray or mist to knock down vapors Vapors knock down water is corrosive or toxic and should be diked for containment. /Hydrogen cyanide, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Preventive Measures :

Evacuation: If material leaking (not on fire) consider evacuation of one-half (1/2) mile radius based on amount of material spilled, location and weather conditions /Hydrogen cyanide, anhydrous, stabilized or hydrocyanic acid, aqueous solution or hydrogen cyanide, liquefied; hydrogen cyanide, anhydrous, stabilized; hydrogen cyanide, anhydrous, stabilized, absorbed in a porous inert material/
[Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, DC: Association of American Railroads, Bureau of Explosives, 1994. 582]**PEER REVIEWED**

Human Toxicity Excerpts :

The excretion of hydrogen cyanide in breath and blood concentrations of cyanide were measured in eight normal subjects. There was no correlation between breath and blood levels of cyanide. Furthermore, breath cyanide concentrations calculated from blood values were much lower than measured values. When saliva was incubated at 37 deg C, hydrogen cyanide was formed in the presence of air but not in a nitrogen atmosphere. No hydrogen cyanide was formed with boiled saliva and the production of hydrogen cyanide from saliva was inhibited by catalase and by 6-N-phopyl-thiouracil. Centrifugation of saliva resulted in a supernatant and a sediment, which were both required for the formation of hydrogen cyanide Dialysis of the supernatant abolished its cyanide forming ability, which could be restored by addition of thiocyanate.
[Lundquist P et al; Arch Toxicol 61 (4): 270-74 (1988)]**PEER REVIEWED**

DOT Emergency Guidelines :

First aid: Move victim to fresh air Call emergency medical care. Apply artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; induce artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Administer oxygen if breathing is difficult Remove and isolate contaminated clothing and shoes In case of contact with substance, immediately flush skin or eyes with running water for at least 20 minutes. Wash skin with soap and water. Keep victim warm and quiet. Effects of exposure (inhalation, ingestion or skin contact) to substance may be delayed. Ensure that medical personnel are aware of the material(s) involved, and take precautions to protect themselves. /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide, Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Protective clothing: Wear positive pressure self-contained breathing apparatus (SCBA). Wear chemical protective clothing which is specifically recommended by the manufacturer. It may provide little or no thermal protection. Structural firefighters' protective clothing is recommended for fire situations only; it is not effective in spill situations /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen

cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide, Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Public safety: Call Emergency Response Telephone Number. ... Isolate spill or leak area immediately for at least 100 to 200 meters (330 to 660 feet) in all directions. Keep unauthorized personnel away. Stay upwind Keep out of low areas. Ventilate closed spaces before entering. /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide; Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Health: Toxic; may be fatal if inhaled, ingested or absorbed through skin Inhalation or contact with some of these materials will irritate or burn skin and eyes. Fire will produce irritating, corrosive and/or toxic gases Vapors may cause dizziness or suffocation. Runoff from fire control or dilution water may cause pollution. /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide; Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Fire or explosion: Highly flammable· Will be easily ignited by heat, sparks or flames Vapors may form explosive mixtures with air. Vapors may travel to source of ignition and flash back. Most vapors are heavier than air. They will spread along ground and collect in low or confined areas (sewers, basements, tanks). Vapor explosion and poison hazard indoors, outdoors or in sewers. Some may polymerize (P) explosively when heated or involved in a fire. Runoff to sewer may create fire or explosion hazard. Containers may explode when heated Many liquids are lighter than water. /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide; Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Evacuation: Spill Fire: If tank, rail car or tank truck is involved in a fire, isolate for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions. /Hydrogen cyanide, anhydrous, stabilized (absorbed), Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide; Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Fire: CAUTION: All these products have a very low flash point. Use of water spray when fighting fire may be inefficient. Small fires: Dry chemical, CO2, water spray or alcohol-resistant foam. Large fires: Water spray, fog or alcohol-resistant foam. Move containers from fire area if you can do it without risk. Dike fire control water for later disposal; do not scatter the material. Do not use straight streams. Fire involving tanks or car/trailer loads: Fight fire from maximum distance or use unmanned hose holders or monitor nozzles. Cool containers with flooding quantities of water until well after fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tank. ALWAYS stay away from the ends of tanks. For massive fire use unmanned hose holders or monitor nozzles; if this is impossible, withdraw from area and let fire burn /Hydrogen cyanide, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide; Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

DOT Emergency Guidelines :

Spill or leak: Fully encapsulating, vapor protective clothing should be worn for spills and leaks with no fire ELIMINATE all ignition sources (no smoking, flares, sparks or flames in immediate area). All equipment used when handling the product must be grounded. Do not touch or walk through spilled material. Stop leak if you can do it without risk. Prevent entry into waterways, sewers, basements or confined areas A vapor suppressing foam may be used to reduce vapors. Small spills: Absorb with earth, sand or other non-combustible material and transfer to containers for later disposal. Use clean non-sparking tools to collect absorbed material. Large spills: Dike far ahead of liquid spill for later disposal Water spray may reduce vapor; but may not prevent ignition in closed spaces. /**Hydrogen cyanide**, anhydrous, stabilized (absorbed); Hydrogen cyanide, solution in alcohol, with not more than 45% Hydrogen cyanide, Hydrogen cyanide, stabilized (absorbed)/
[U.S. Department of Transportation. 1996 North American Emergency Response Guidebook. A Guidebook for First Responders During the Initial Phase of aHazardous Materials/Dangerous Goods Incident. U.S. Department of Transportation (U.S. DOT) Research and Special Programs Administration, Office of HazardousMaterials Initiatives and Training (DHM-50), Washington, D.C. (1996).,p. G-131]**PEER REVIEWED**

Environmental Fate :

ATMOSPHERIC FATE: The reaction of **hydrogen cyanide** with photochemically generated hydroxyl radicals proceeds fairly slowly Based on a reaction rate constant of 3x10-14 cu m/(molecules-sec) at 25 deg C, and assuming an ambient hydroxyl radical concentration of 8x10+5 molecules/cu m, the half-life for the reaction of hydrogen cyanide vapor with hydroxyl radicals in the atmosphere has been approximately 334 days. Hydrogen cyanide is expected to be resistent to direct photolysis The relatively slow rate of degradation of hydrogen cyanide suggests that this compound has the potential to be transported over long distances before being removed by physical or chemical processes. Since hydrogen cyanide is miscible in water, it appears that wet deposition may be an important fate process. Metal cyanide particles are expected to be removed from air by both wet and dry deposition.
[DHHS/ATSDR; Toxicological Profile for Cyanide (Draft) p.76 (1/88)]**PEER REVIEWED**

Non-Human Toxicity Excerpts :

HEALTH AND SAFETY PLAN

Field Activities Covered Under This Plan:

Task	Description	Type	Level of Protection: Primary	Level of Protection: Contingency	Date of Activities
1	Document field activities	☐ Intrusive ☒ Nonintrusive	☐ C ☒ D ☐ Modified	☐ C ☐ D ☒ Modified	6/04/01 to ?
2	Provide contractor oversight	☐ Intrusive ☒ Nonintrusive	☐ C ☒ D ☐ Modified	☐ C ☐ D ☒ Modified	6/04/01 to ?

Site Personnel and Responsibilities (include subcontractors):

Employee Name and Office Code	Task	Responsibilities
David Sawicki	1 &2	Project Manager or Field Team Leader: Directs project investigation activities, makes site safety coordinator (SSC) aware of pertinent project developments and plans, and maintains communications with client as necessary.
David Sawicki	1 &2	Site Safety Coordinator (SSC): Ensures that appropriate personal protective equipment (PPE) is available, enforces proper utilization of PPE by on-site personnel, suspends investigative work if he or she believes that site personnel are or may be exposed to an immediate health hazard, implements the health and safety plan, and reports any observed deviations from anticipated conditions described in the health and safety plan to the health and safety representative.
David Sawicki	1 &2	Field Personnel: Complete tasks as directed by the project manager, field team leader, and SSC and follow all procedures and guidelines established in the Tetra Tech, Inc., Health and Safety Manual.

HEALTH AND SAFETY PLAN

Protective Equipment: (Indicate type or material as necessary for each task; attach additional sheets as necessary)

Task: [X] 1 [x] 2	Task: [ ] 1 [ ] 2
Level: [ ] C [x] D [ ] A or B	Level: [ ] C [ ] D [ ] A or B
[x] Primary [ ] Contingency	[ ] Primary [ ] Contingency
RESPIRATORY	**RESPIRATORY**
[x] Not needed	[ ] Not needed
[ ] APR: _____	[ ] APR: _____
[ ] Cartridge: _____	[ ] Cartridge: _____
[ ] Escape mask: _____	[ ] Escape mask: _____
[ ] Other: _____	[ ] Other: _____
PROTECTIVE CLOTHING	**PROTECTIVE CLOTHING**
[x] Not needed	[ ] Not needed
[ ] Tyvek® coveralls: _____	[ ] Tyvek® coveralls: _____
[ ] Saranex® coveralls: _____	[ ] Saranex® coveralls: _____
[ ] Coveralls: _____	[ ] Coveralls: _____
[ ] Other: _____	[ ] Other: _____
HEAD AND EYE	**HEAD AND EYE**
[ ] Not needed	[ ] Not needed
[x] Safety glasses: _____	[ ] Safety glasses: _____
[ ] Face shield: _____	[ ] Face shield: _____
[ ] Goggles: _____	[ ] Goggles: _____
[X] Hard hat: _____	[ ] Hard hat: _____
[ ] Other: _____	[ ] Other: _____
GLOVES	**GLOVES**
[x] Not needed	[ ] Not needed
[ ] Undergloves: _____	[ ] Undergloves: _____
[ ] Gloves: _____	[ ] Gloves: _____
[ ] Overgloves: _____	[ ] Overgloves: _____
FIRST AID EQUIPMENT	**FIRST AID EQUIPMENT**
[ ] Not needed	[ ] Not needed
[X] Standard First Aid kit	[X] Standard First Aid kit
[ ] Portable eyewash	[ ] Portable eyewash
BOOTS	**BOOTS**
[ ] Not needed	[ ] Not needed
[X] Work boots: Steel-Toe/Steel Shank	[X] Work boots: Steel-Toe/ Steel Shank
[x] Overboots: booties	[ ] Overboots: _____
OTHER *(specify)* _____	OTHER *(specify)* _____

Note: APR = Air purifying respirator

Monitoring Equipment: (Specify instruments needed for each task; attach additional sheets as necessary)

Instrument	Task	Instrument Reading	Action Guideline	Comments
Combustible gas indicator model	☐ 1 ☐ 2	0 to 10% LEL	No explosion hazard	☒ Not needed
		10 to 25% LEL	Potential explosion hazard; notify SSC	
		>25% LEL	Explosion hazard; interrupt task, evacuate site; notify SSC	
O_2 meter model:	☐ 1 ☐ 2	>23.5% O_2	Potential fire hazard; evacuate site	☒ Not needed
		23 5 to 19 5% O_2	Oxygen level normal	
		<19.5% O_2	Oxygen deficiency, interrupt task; evacuate site, notify SSC	
Radiation survey meter model:	☐ 1 ☐ 2	<2 mrem per hour	Normal background	Note: Annual exposure not to exceed 1,250 mrem per quarter ☒ Not needed
		Three times background	Notify SSC	
		>2 mrem per hour	Radiological hazard, interrupt task; evacuate site; notify SSC	
Photoionization detector model: ☐ 11 7 eV ☒ 10.2 eV ☐ 9.8 eV ☐ _____ eV	☒ 1 ☒ 2	>0 to 5 ppm above background	Level D	☐ Not needed
		>5 to 20 ppm above background	Level C	
		>20 ppm above background	Evacuate site, notify SSC	
Flame ionization detector model	☐ 1 ☐ 2	>0 to 5 ppm above background	Level D	☒ Not needed
		>5 to 20 ppm above background	Level C	
		>20 ppm above background	Evacuate site, notify SSC	
Detector tube models:	☐ 1 ☐ 2	Specify	Specify	Note: The action level for upgrading the level of protection is one-half of the contaminant's PEL. If the PEL is reached, evacuate the site and notify the SSC ☒ Not needed
Respirable dust monitor model:	☐ 1 ☐ 2	Specify	Specify	☒ Not needed
Other (specify)	☐ 1 ☐ 2	Specify	Specify	☒ Not needed

Notes: eV = Electron volt LEL = Lower explosive limit mrem = Millirem O_2 = Oxygen PEL = Permissible exposure limit ppm = Part per million

Additional Comments:	Emergency Contacts:	Telephone
	U.S. Coast Guard National Response Center	800/424-8801
	InfoTrac	800/535-5053
	Fire department	911 or _____
	Police department	911 or _____
	Tetra Tech EM Inc. Personnel:	
	Human Resource Development: Norman Endlich	703/390-0626
	Health & Safety Representative: Judith Wagner	847/255-4166
	Office Health and Safety Coordinator:	
	Project Manager: Dave sawicki	
	Site Safety Coordinator:	

Personnel Decontamination and Disposal Method:	Medical Emergency:
Personnel will follow the U.S. Environmental Protection Agency's "Standard Operating Safety Guides" for decontamination procedures for modified Level D personal protection (with modified Level C contingency). The following decontamination stations should be set up in each decontamination zone: • Segregated equipment drop • Boot and glove wash and rinse • Disposable glove, bootie, and coverall removal and segregation station • Safety glasses and hard hat removal station • Hand and face wash and rinse If site conditions require upgrade to Level C, a station must be set up for respirator removal, respirator decontamination, and cartridge disposal. All disposable equipment, clothing, and wash water will be double-bagged or containerized in an acceptable manner and disposed of in accordance with local regulations.	Hospital Name: Henry Ford Hospital Address:2799 w. grand blvd. Detroit, MI 48202 Hospital Telephone: Emergency - _____911 General : 313-916-2600 Ambulance Telephone: 911 or _____ Route to Hospital: (see Page 10 of 12 for route map)

Note: This page must be posted on site.

YAHOO! YELLOW PAGES

Welcome, Guest User

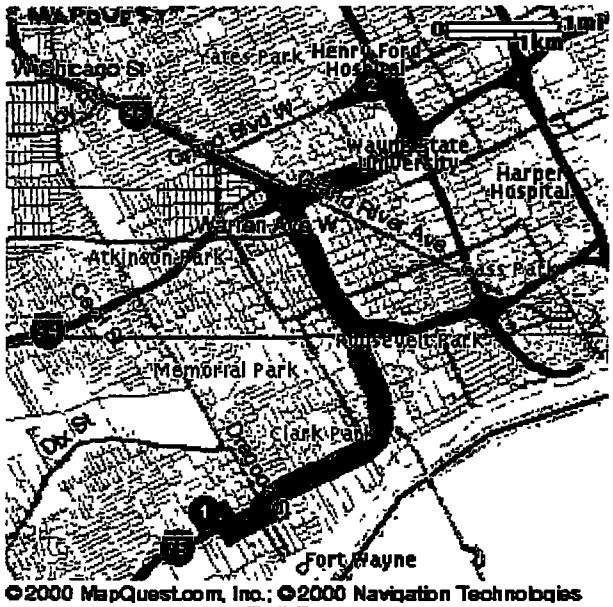
Yahoo! Yellow Pages

Starting from: 301 s green road, Detroit, MI 48209

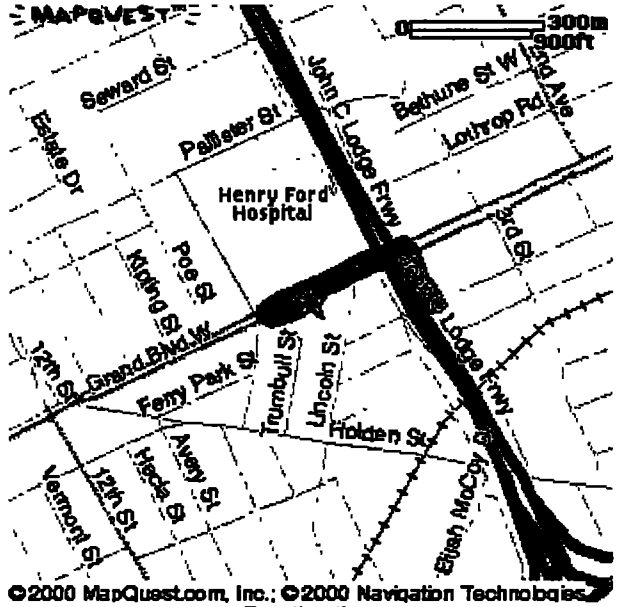
Arriving at: Henry Ford Hospital & Medical
2799 W Grand Blvd, Detroit, MI 48202
(313) 916-2600

Distance: 6 6 miles **Approximate Travel Time:** 9 mins

- Email Directions
- Get Reverse Directions
- Text Only Driving Directions



Full Route



Destination

	Directions	Miles
1.	Start out going Southeast on **BEARD ST** towards **LEXINGTON ST** by turning left.	0.1
2.	Turn LEFT onto **LAFAYETTE BLVD W**.	0.1
3.	Turn RIGHT onto **WATERMAN ST**	0.2
4.	Turn LEFT onto **FORT ST W/MI-3**	0.4
5.	Turn LEFT onto **DRAGOON ST**.	0.1
6.	Turn RIGHT onto **FISHER FRWY W**.	0 0
7. .	Turn SLIGHT LEFT to take the **I-75 NORTH** ramp.	0.1
8.	Merge onto **I-75 N**	1 1
9	Take the **I-96 WEST** exit, exit number **48**, on the left towards **LANSING**.	1.3
10.	Merge onto **I-96 W/JEFFRIES FRWY**.	0.8
11.	Take the **I-94** exit, exit number **190A**, towards **CHICAGO/PORT HURON**.	0 1
12	Merge onto **I-94 EAST RAMP**.	0.2

13	Merge onto **I-94 E**	1 0
14	Take the **M-10 NORTH** exit, exit number **215B**, on the left	0 4
15.	Merge onto **JOHN C LODGE FRWY/MI-10 N**	0.2
16	Take the **MILWAUKEE AVE** exit towards **W GRAND BLVD**	0.1
17	Keep LEFT at the fork in the ramp	0.0
18.	Stay straight to go onto **JOHN C LODGE FRWY**	0.1
19.	Turn LEFT onto **W GENERAL MOTORS BLVD/GRAND BLVD W**	0.3

When using any driving directions or map, it's a good idea to do a reality check and make sure the road still exists, watch out for construction, and follow all traffic safety precautions. This is only to be used as an aid in planning.

Driving Directions New Location

1 **Enter a starting address** or select from My Locations

My Locations [ ] Sign In

(Address, Intersection or Airport Code)

Address 301 s. green road

City, State or Zip Detroit, MI 48209

Country United States

2 **Enter a destination address** or select from My Locations

My Locations [ ] Sign In

(Address, Intersection or Airport Code)

Address 2799 W Grand Blvd

City, State or Zip Detroit, MI 48202

Country United States

Get Directions

APPROVAL AND SIGN-OFF FORM

Project No. _____

I have read, understood, and agree with the information set forth in this Health and Safety Plan and will follow the direction of the Site Safety Coordinator as well as procedures and guidelines established in the Tetra Tech, Inc., Health and Safety Manual. I understand the training and medical requirements for conducting field work and have met these requirements.

Name	Signature	Date
DAVID SAWICKI		6/01/01

APPROVALS: *(Two Signatures Required)*

Site Safety Coordinator	Date: 6/01/01
Health and Safety Representative or Designee	Date

DEFINITIONS

Intrusive - Work involving excavation to any depth, drilling, opening of monitoring wells, most sampling, and Geoprobe® work

Nonintrusive - Generally refers to site walk-throughs or field reconnaissance

Levels of Protection

Modified Level D - Hard hat, safety boots, and glasses

Level D - Items listed for modified Level D above, **PLUS** protective clothing such as gloves, boot covers, and Tyvek® or Saranex® coveralls

Modified Level C - Hard hat, safety boots, glasses, and air purifying respirators with appropriate cartridges

Level C - Items listed for modified Level C above, **PLUS** protective clothing such as gloves, boot covers, and Tyvek® or Saranex® coveralls

Emergency Contacts

InfoTrac - For issues related to incidents involving the transportation of hazardous chemicals; this hotline provides accident assistance 24 hours per day, 7 days per week

U.S. Coast Guard National Response Center - For issues related to spill containment, cleanup, and damage assessment; this hotline will direct spill information to the appropriate state or region